

15

OBSERVATIONS UPON A CURIOUS AND NOT
UNCOMMON FORM OF EXTREME ACCELERATION
OF THE AURICLE. "AURICULAR FLUTTER."

By THOMAS LEWIS.

Reprinted from "Heart."
Vol. IV, No. 2, November 30, 1912.

WELLCOME INSTITUTE LIBRARY	
Coll.	welMOmec
Coll.	pam
No.	WG 200
	1 9 1 2
	L 6 7 0



22500892576

[Reprinted from "Heart."
Vol. IV, No. 2, November 30, 1912.]

OBSERVATIONS UPON A CURIOUS AND NOT UNCOMMON FORM
OF EXTREME ACCELERATION OF THE AURICLE.
"AURICULAR FLUTTER."

BY THOMAS LEWIS.*

(Cardiographic Department, University College Hospital
Medical School).

Introductory Remarks.

FOUR years ago Hertz and Goodhart¹ published the notes of a case in which it was evident that the auricles had assumed a very rapid action. The case was one in which the auricles contracted at a rate varying between 216 and 234 per minute, while the ventricular action was slow. Isolated cases of a similar nature have since been recorded by Jolly and Ritchie,² by Rihl,⁹ and by myself.⁷ The condition has been regarded by those interested in the recent progress of cardiac investigation as a rare affection.

While patients in whom a slow or irregular ventricular action have been more and more extensively investigated by means of graphic methods, the necessity for a routine examination of simple cardiac accelerations in the same manner has not been recognised.

It appears, as I hope to show in the present communication, that extreme acceleration of the auricular contractions to a rate of 300 per minute is not an infrequent human malady; and that the combined picture of auricular acceleration and heart-block is relatively common. It will also become apparent that if the routine examination of patients who present rapid heart action by modern methods, and especially by electrocardiographic means, is not undertaken, the condition will often escape detection.

A series of cases which has recently come under the observation of the writer has clearly shown, not only that such examination is imperative if the true nature of a given tachycardia is to be ascertained, for such cases may readily pass for other and more simple forms of acceleration, but that a detailed acquaintanceship with the forms which the electrocardiographic curve may take is essential. In most instances there are few or no signs of the extreme auricular acceleration, other than those found in the electrocardiographic pictures; and in many of these, the degree of acceleration is by no means obvious if such curves are hastily scanned. The reason for the obscurity of the signs is twofold, for, in the first instance, the auricular contraction rate is usually double the ventricular, and alternate auricular systoles are buried in the ventricular systole and pass unnoticed; and, in the second instance, the systoles of the auricles have little mechanical effect upon the blood content of either veins or ventricles, and the movements of the auricular walls are small.

* Working under the tenure of a Beit Memorial Fellowship.

That acceleration of a regularly contracting auricle to a rate of 300 and even 330 per minute has frequently remained unsuspected in the past, may seem remarkable, but nothing is more certain than that such is the case and that the auricles may pursue this hurried movement so covertly that none of the older methods of examination can discover it.

It is my purpose in the present communication to show that the condition is relatively common; to point out the signs, be they arterial, venous or electric by which the malady, as it commonly presents itself to our notice, may be recognised; and further, to describe it as an affection which must stand, for clinical if not for pathological purposes, in a category of its own. The need for isolating it in this manner from other disturbances of heart mechanism is impressed by its special relations to certain forms of abnormal heart action, by the peculiar course which it pursues and by the curious manner in which it reacts to remedies.

For the reasons stated, it seems expedient that the disorder should be regarded as a distinct type, and that it should be treated separately even though complete separation may not be possible from new rhythms which, though of auricular origin, are of lower grade. The readiest means of isolating it in this manner is to apply to it a distinctive name, and there seems none better than that adopted by Jolly and Ritchie in the description of their case, namely, "auricular flutter"; for this term aptly describes the essential condition, it avoids reference to the varying grades of ventricular response and clearly serves to distinguish it from the allied disorder, "auricular fibrillation." The distinction from tachycardia of lower rate, though also of auricular origin, is more arbitrary, though I think essential at the present time. The auricular rate may be increased to any figure intermediate between 72 and 335 beats per minute; but there are certain features which accelerations of over 200 appear to hold in common; and so, even if the distinction subsequently proves not to be strictly valid on the pathological side, the separation is not without merit from the clinical standpoint.

In the following pages, I propose to deal in the first place with my own observations; secondly to summarise former observations and to revise certain previous reports; and, finally, to discuss in a general account the disorder as a clinical entity.

Observations.

CASE 1. M., a married woman, aged 50, came under observation on April the 19th, 1912.

Past history. There had been six children; one child died at three months of age of "rheumatic fever." The patient was affected by rheumatic fever herself when this child was born; she was then 23 years of age.

History of cardiac condition. She had suffered from shortness of breath for eleven years. For four months it had been more severe, being especially noticeable after exertion. Palpitation had been present for four months, and there was also some pain in the precordial region. Her feet had never swollen; there had never been hæmoptysis. Two months before admission, while lifting a heavy saucepan, she had a feeling "as if her heart was displaced"; she experienced great breathlessness and palpitation at the time and took to bed. It was the first attack of its kind.

Condition, April the 19th, 1912. A stout woman, who showed a trace of cyanosis. Breathlessness was obvious while she lay flat in bed. There was no dropsy. The liver dulness came an inch below the costal margin; the organ was readily felt, and there was tenderness to pressure over it. The urine was normal. During her stay she had fever for a few days (97-101 degrees Fahr.) and the ankle and wrist joints became swollen and painful.

The limits of cardiac dulness lay 2 and 5 inches to the right and left of the mid-sternal line. A short systolic murmur was heard at the apex. The heart sounds were otherwise normal. At the time of observation, she was upon thirty minims of the tincture of digitalis and she had had in all seven and a half drachms of this drug or its equivalent. The ventricular rate was usually 160, though there were periods of irregular and slower action from time to time. The rate of the heart beat was not affected by posture. The observations upon the heart mechanism and the course of the condition are summarised in the accompanying table.

DATE	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
3-4-12	Pulse counts	160-118 -160		Tinct. Digit.	Date of admission.
19-4-12	Electrocardiographic	156	312	Tinct. Digit. m. xxx	Chiefly 2 : 1. Also irregular periods of 2 : 1 and 3 : 1.
20-4-12 to 25-4-12	Pulse counts.	120-158		m. xxx daily till the 2nd	Usually regular; some irregular periods. Fever 101° and rheumatism of foot. Nausea.
2-5-12	Electrocardiographic	123-160	320	m. xlv.	Chiefly 2 : 1; also irregular periods of 2 : 1 and 3 : 1.
4-5-12	Electrocardiographic	81	324	m. lx	4 : 1 heart-block; regular.
6-5-12	Electrocardiographic	79		m. lx (Total dr. xviii)	Fibrillation of the auricles. Digitalis stopped.
9-5-12	Pulse counts	72-96		None	Fibrillation still.
10-5-12	Pulse counts	88-92		None	Fibrillation still.
12-5-12	Pulse counts	82-90		None	Pulse became regular. Probably returned to normal rhythm.
13-5-12	Electrocardiographic	81	81	None	Pulse regular, normal rhythm.
15-5-12*	Pulse counts	72-92		None	Pulse regular, normal rhythm.
29-5-12	Electrocardiographic	108	108	None	Pulse regular, normal rhythm.
1-6-12	Electrocardiographic	91	91	None	Pulse regular, normal rhythm.
10-6-12	Pulse counts	70-88			Normal rhythm still. The breathlessness has gone. The liver has receded beneath the ribs. There is no pain or palpitation. She feels much stronger and her colour is natural.
10-6-12	Discharged from hospital.				
7-8-12	Electrocardiographic	80-90	80-90	None	Normal rhythm. Rate rise to 101 on standing and falls to 80 lying. Slight dropsy of ankles and a little shortness of breath.

* On the 15th of May, in the early morning, she had a slight stroke; consciousness was not lost but the left half of the face and left arm became paralysed and speech was impaired. The paresis cleared up entirely during the course of the next few days.

Fig. 19 is illustrative of the electrocardiograms taken during the early phases of the patient's treatment. The initial ventricular peaks are clearly defined in all leads; *T* is seen in lead *I* and slightly in lead *II*; in lead *III* it cannot be found. It is difficult to identify auricular representatives in lead *I*. In lead *II* they appear as upward deflections; the *P-R* interval seems to have a duration of about .1 sec.. In lead *III*, the shape of *P* is more complex; its general outline may be described as convex,* the upstroke being more abrupt than the downstroke which is broken by a notch. The auricular curves are contiguous one with another, the line is never written horizontally. The auricular systoles run through the ventricular and are twice as numerous as the latter. Both chambers beat with perfect regularity, the auricles at 320 and the ventricle at 160.

Fig. 20 represents the action of the heart two days later; while under the action of digitalis heart-block had increased. In this figure the shapes of the auricular summits and their relation to each other and the ventricular systoles is more clearly seen. Both chambers still beat quite regularly but the ventricle is now contracting but a fourth as rapidly as the auricle (auricular rate 324, ventricular 81); each fourth auricular summit is buried in the opening phases of ventricular systole. The general form of both auricular and ventricular electric curves has been maintained (see Fig. 19).

Fig. 21 was taken two days later, when the auricles were fibrillating under the influence of digitalis. The ventricular action is slow (the rate was 79) but quite irregular; the regular auricular summits have given place to smaller and irregular oscillations which are most clearly seen in lead *III* (*f.f.*).

Fig. 22 shows the perfectly normal mechanism, which became established shortly after the digitalis was omitted. There is no sign of rapid and continuous oscillations in the curves; a single auricular complex occurs before each ventricular systole. The heart is beating perfectly regularly and slowly.

The series of curves is a very complete one. We have the electrocardiograms from the same heart, beating in three completely different fashions. The auricular representatives of the normal period are quite unlike those of the period of flutter. In all, the shape of the ventricular complexes is maintained in the separate leads, demonstrating quite clearly that in each instance the ventricular beats were propagated from the auricles.

Summary. In a woman of 50 years, an attack of rapid heart action was observed which had a probable duration of three months. The rate of the auricular contractions surpassed 300 per minute, the highest count being 324 per minute; the ventricle usually responded to alternate auricular contractions. Under high doses of digitalis the ventricular rate fell to a fourth of the auricular rate, the latter being maintained. Eventually and

* The reasons for this statement are given under *CASE 3*.

TABLE OF ATTACKS IN CASE 2, COMPILED FROM A DIARY AND SHOWING THE NUMBER OF ATTACKS AND THEIR DURATION, OVER A PERIOD OF TEN YEARS.

YEAR	1902	1903	1904	1905	1906	1907	1908	1909	1910	1911	Total Number.
January	3 (17)*	3 (21)	2 (20)	5 (19)	5 (58)	2 (23)	3 (36)	4 (19)	8 (23)	5 (191)	40
February	3 (3)	1 (8)	2 (10)	4 (26)	5 (40)	8 (51)	5 (8)	10 (26)	3 (26)	1? (240)	42
March	3 (16)	5 (60)	2 (7)	2 (24)	5 (12)	4 (19)	8 (36)	4 (27)	6 (19)	0	39
April	3 (13)	2 (16)	2 (8)	4 (49)	6 (28)	7 (45)	3 (48)	4 (9)	5 (18)	1 (6)	37
May	4 (5)	3 (9)	1 (13)	1 (14)	13 (63)	3 (39)	3 (14)	8 (31)	3 (3)	5 (51)	44
June	0	3 (14)	4 (29)	4 (15)	3 (11)	6 (11)	2 (4)	4 (16)	6 (9)	3 (26)	35
July	5 (31)	3 (4)	3 (6)	2 (8)	5 (20)	4 (23)	7 (13)	10 (37)	4 (31)	2 (21)	45
August	1 (7)	4 (21)	2 (1)	1? (22)	9 ?	2 (8)	6 (13)	2 (4)	6 (8)	1 (4)	34
September	4 (25)	4 (26)	5 (14)	3 (27)	5 (20)	5 (39)	7 (42)	5 (8)	5 (23)	3 (24)	46
October	2 (31)	4 (16)	2 (22)	5 (59)	6 (43)	3 (27)	2 (18)	8 (28)	3 (23)	10 (56)	45
November	1 (10)	5 (38)	5 (47)	4 (34)	5 (62)	5 (55)	10 (66)	8 (22)	7 (46)	1 (16)	51
December	3 (49)	1 (14)	3 (25)	3 (7)	5 (64)	5 (38)	1 (10)	4 (16)	5 (17)		30
TOTALS	32 (207)	38 (243)	33 (202)	38 (304)	72 (401+)	54 (378)	57 (296)	71 (243)	61 (246)	32 (634)	488

* The first figure gives the number of attacks, the second gives the total duration in hours (minutes omitted).

under the influence of the drug, the auricles fibrillated ; the digitalis was then withdrawn and in a few days the normal rhythm was restored and was maintained so long as the patient was under observation. Her symptoms were considerably relieved.

CASE 2. E. G., a clergyman, aged 65, came under continuous observation on the 2nd of February, 1912.

Past illnesses. He suffered from scarlet fever at 5 and 13 years of age ; on the second occasion the fever was followed by osteomyelitis of the left tibia and he was in bed for six months. At 15 he had congestion of the lungs ; and at 52 pleurisy. For three years he had been afflicted with a prostatic enlargement and had used a catheter thrice daily for three months.

History of heart condition. For thirty years he had been the subject of attacks of tachycardia. The first attack, at the age of 35, lasted several hours and laid him up ; his doctor told him his heart was normal except for its excessive rate. Six years later, he had his second attack, and was attended for it by Dr. Bengafeld, of Edmonton, who writes to me " I quite well remember him as being subject to severe attacks of tachycardia, which I could not account for. The pulse rate would go up to fully 140 or 150." From the age of 41 he had attacks each six months or each year which lasted from four to twenty hours. The attacks were accompanied by shortness of breath, a sense of precordial oppression and a feeling as if a pendulum were swinging fast in the chest. There were no anginal symptoms. The longest attacks exhausted him very much, but the shorter ones left him perfectly fit a few minutes after their cessation. He almost always continued his work if they came on while he was occupied. He often suffered from flatulence during the paroxysms. At the age of 50 he had a great deal of trouble in his parish and was much upset by it. The attacks became more frequent, commencing each week or each day. For the preceding ten years he had kept a complete diary of the attacks. From January, 1902, until November, 1912, there were 489 attacks at least. The average duration was about six hours, the shortest attack lasted a few minutes and the longest ten days. The distribution of the attacks over the ten years is seen in the table of monthly incidence on the last page. There is surprising uniformity in the distribution of the paroxysms over this period, and the totals for the several months show no seasonal incidence. A chart (page 178) of the frequency of onset during the several hours of the day and night shows the greatest frequency in the early hours of the morning, early afternoon and late evening. He attributed many of the attacks to excitement and stated that the evening attacks often followed his Sunday's sermon. He believed that meals had no relation to the attacks. The tachycardia became continuous in December, 1912, and was maintained until May, 1912.

Condition, 6th of May, 1912. A frail and tremulous subject. The remaining teeth were carious. There was enlargement of the prostate and infection of the bladder. The heart limits were normal, the ventricular rate was regular at 130, the sounds were normal. The arm arteries were soft ; the pulse tension was not increased. Radiographic examination showed dilatation of the aorta. The auricular movements could not be seen.

The heart was examined polygraphically and electrocardiographically on a number of occasions. The general result and course may be stated in tabular fashion.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
29-2-12	Electrocardiographic	138	276		2 : 1 block.
26-3-12	Electrocardiographic	96+	289		3 : 1 block chiefly. Ventricle a little irregular ; 2 : 1 periods present.
2-4-12	Electrocardiographic	138	276		2 : 1 block.
2-5-12	Electrocardiographic	133	266		2 : 1 block.
3 ^{to} 7-5-12	Pulse counts	96-140			
8-5-12	Polygraphic	94		Tinct. Digit. m. xxx	Probably 2 : 1 and 3 : 1 mixed.

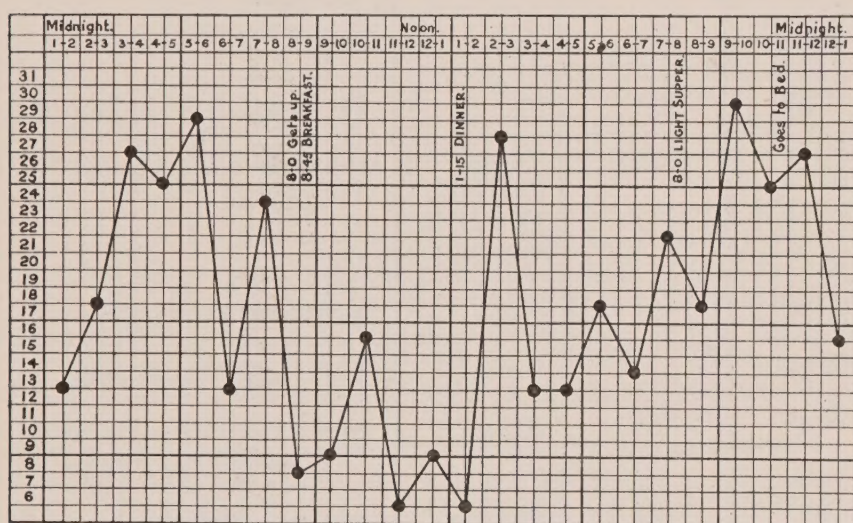
DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
9-5-12	Pulse counts	108-112		Tinct. Digit m. xxx	
10-5-12	Pulse counts	100-120		m. xxx	
11-5-12	Polygraphic	92	? 276	m. xxxv	Probably 3 : 1 block
12-5-12	Electrocardiographic	- 137	274	m. xlv	2 : 1 block. Occasional 3 : 1 and 4 : 1 periods in other curves.
13-5-12	Pulse counts	76-108		m. l *	
14-5-12	Pulse counts	70-92		m. lxv	
15-5-12	Pulse counts	76-96		m. lxxv	} Probably 4 : 1. } Pulse regular.
16-5-12	Pulse counts	72-96		m. c	
17-5-12	Electrocardiographic	58		m. l	Fibrillation of the auricles.
		152	? 150	Digit. stopped. (Total dr. viii, m. v.)	Regular tachycardia. Vomiting.
18-5-12	Pulse counts	48-64		None	Auricular fibrillation.
19-5-12	Pulse counts	62-68		None	Auricular fibrillation.
20-5-12	Polygraphic	64	64	None	Normal rhythm. Occasion- al premature beats.
21-5-12	Electrocardiographic	92	92	None	Normal rhythm.
22-5-12	Polygraphic	54-66	54-66	None	Normal rhythm.
23-5-12	Passed from observation. The general condition is much improved. His breathing is fairly easy and he feels in better health.				
31-5-12	Patient writes that his pulse continues to be normal.				
6-6-12	Electrocardiographic	102	102		Normal rhythm. Has had a few short attacks. Condition of patient greatly improved.
27-6-12	Electrocardiographic	84	84		The patient states he has had one or two short attacks of a few minutes and one long one since he was last seen. His condition shows steady improvement.

* Pressure upon the right or left vagus while this patient was under digitalis, produced additional slowing of the ventricle.

Fig. 23 is one of the earliest electrocardiograms taken from this patient. The auricular rate is 289 per minute. The *P* summits in lead *I* are not seen. In leads *II* and *III* they form, together, continuous wavy lines, the individual waves of which are placed regularly. It is not certain where each auricular representative starts or finishes, though it is probable, from a consideration of similar curves in other cases, that each is

convex and starts with the upstroke. The ventricular action is not quite regular and the beats have not always the same relation to the auricular representatives with which they fall; for the most the curve shows 3:1 heart-block and probably consists as a whole of 3:1 and 2:1 periods. *T* is seen to some extent in lead *I* only.

Fig. 24 was taken on the 12th of May while the patient was beginning to be influenced by digitalis. The auricular curves are similar to those of the preceding figure (rate 274), but the ventricular curves are relatively more numerous. The curve shows 2:1 heart-block for the most part; there are occasional periods of 3:1 and 4:1 block. Five days later the heart action became grossly irregular in response to fuller doses of the drug. Curves of this day are shown in Fig. 25 and 26.



A Chart showing the times of onset of 428 attacks. Compiled from a diary kept for ten years. (CASE 2, p. 176).

Fig. 25 shows a slow and completely irregular ventricular action (rate 58). The auricular waves are no longer present. *T* is clearly seen in all leads, and following the *Q*, *R* and *S* group in leads *II* and *III*, there are extra deflections from time to time, which are not understood. These curves in all probability represent auricular fibrillation, the oscillations being too small

to record. From time to time during the examination, the heart's action became rapid and perfectly regular at 152 per minute. The electrocardiograms are shown in Fig. 26. The type of the ventricular complexes is still maintained, though in leads *II* and *III* the curious deflections following the *Q*, *R* and *S* group are constantly present. No trace of auricular summits can be found; the exact meaning of this mechanism is unknown, but it probably results from simultaneous contraction in auricle and ventricle. A few days later, after the digitalis had been omitted, the normal mechanism became restored; it is illustrated by Fig. 27. Here each regular ventricular contraction is preceded by a single auricular contraction of normal type.

This series of curves is a very complete one. Four perfectly distinct mechanisms may be compared in electrocardiograms from the same patient. The auricular representatives of the normal period are unlike those of the period of flutter. The ventricular complexes maintain their general form throughout the opening phases in all; for the impulses in all are generated above the ventricle.

Summary. A man of 65 years had been the subject of attacks of tachycardia for 30 years (rate 140-150). Eventually the condition of rapid

heart action became established; it lasted for a period of five months; during this time the auricles were beating at a rate of approximately 280 per minute, while the ventricles responded as a rule to alternate auricular impulses. Under large doses of digitalis, and after a preliminary period in which higher grades of heart-block were seen, the auricles passed into fibrillation; the drug being withdrawn the normal rhythm was restored and was maintained so long as the patient was under observation.* While the auricular flutter was present, the rate of contractions was uninfluenced by posture, and by digitalis administration. As a result of treatment his general condition improved very considerably.

CASE 3. E. B., a clergyman, aged 53, came under continuous observation on May the 14th, 1912.

Past illnesses. He suffered from the usual children's ailments. At the age of 41 he had diphtheria and it was followed by palatal paralysis and weakness of the limbs and eye muscles. Apart from these illnesses and his heart condition, he had always been a healthy man; hard worked and athletic, he had led a strenuous life.

History of heart condition. His attacks of palpitation started at the age of 16 when he was at school. The first attack came on after a hard game of football. For several years he had two or three attacks following exertion; they always started abruptly without warning and ended with equal suddenness. The attacks became more frequent when he was 20, occurring four or five times a year; excitement and exposure to cold induced them; they usually lasted twenty minutes, the longest attack was of about two hours duration. The heart action was very rapid. Following upon the diphtheria they became more frequent and more severe, exhausting him and interfering with his work. He had to change his parish, seeking lighter work.

Three years before he came under observation an attack came on after a long cycle ride, and it continued until he sought advice. During this period his pulse rate lay continuously between 140 and 160; he had been examined on numerous occasions and was under medical supervision for long periods; his chief symptoms were disinclination to exert himself and easy fatigue. These sensations and consciousness of the heart's action alone disturbed him.

Condition on May the 14th, 1912. A sturdy and robust man of good colour, he was a little tremulous. He showed anxiety, but this was apparently the result of his inability to follow his occupation rather than fear for his condition. The right and left limits of cardiac dulness lay 0 and 4 inches to the right and left of the mid-sternal line respectively. The heart sounds were natural, and there were no murmurs. The arteries presented no signs of thickening, the blood pressure was normal; the urine was normal. The heart's action was regular but continuously rapid.

He was seen on two occasions prior to coming under continuous observation and on both the heart's mechanism was similar. A synopsis of observations is given in the accompanying table.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
10-1-12	Electrocardiographic	162	324	None	2:1 heart-block. Auricular rate constant after exertion and after exercise.
3-5-12	Electrocardiographic	156	312	None	2:1 heart-block. Auricular rate constant after exertion and after exercise.
14-5-12	Polygraphic	135-147	294	None	2:1 with occasional 3:1 and 4:1 periods. (Patient has been under digitalis recently).
15-5-12	Electrocardiographic	156	312	Nativ.Digit Gran. i	

* In July the patient decided upon an operation for his enlarged prostate; he was a good deal worried about it and he had a return of the flutter. The operation was performed, but he died within eight days. A post-mortem could not be obtained.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
16-5-12	Electrocardiographic	106	308	Digit. Gran. i	2 : 1, 3 : 1 and 5 : 1 irregular. Auricular rate constant after exertion and after resting.
17-5-12 and 18-5-12	Pulse counts	70-100		ii	Very irregular.
				ii	Very irregular.
19-5-12	Polygraphic	85-140	280	ii	2 : 1 with very frequent 3 : 1 and 4 : 1 periods (irregular). (Ventricle slower while resting).
20-5-12	Polygraphic	84	300	iii	Very irregular. 2 : 1, 3 : 1, 4 : 1 and 5 : 1 mixed. (Ventricle slow while resting).
21-5-12	Electrocardiographic	75	288	iii	4 : 1 and occasional 2 : 1. (Ventricle slow while resting).
22-5-12	Pulse counts	70-90		iii	Very irregular.
23-5-12	Electrocardiographic (after exercise)	139	278	iii	2 : 1 after exercise. Slow and irregular while resting.
24-5-12	Polygraphic	70	301	iii	Very irregular. 3 : 1, 4 : 1 and 5 : 1 mixed.
25-5-12	Polygraphic	114	296	iv	2 : 1, 3 : 1 and 4 : 1 mixed. Very irregular.
26-5-12	Polygraphic	92	264	iv	Very irregular. 2 : 1, 3 : 1, 4 : 1 and 5 : 1 mixed.
27-5-12	Pulse counts	90-140		iv	Pulse slow and very irregular while resting. Fast and regular while standing.
28-5-12*	Polygraphic	74	280	i Granules stopped, (total 36)	3 : 1, 4 : 1 and 5 : 1; irregular.

*On the 28th the patient experienced nausea and retching. There had been yellow vision and increased sensibility to auditory and touch impressions for several days. The digitalis administration had consequently to be abandoned. It was decided to try strophanthus after a few days of rest.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
29-5-12	Polygraphic	73	288	None	3 : 1, 4 : 1 and 5 : 1 periods mixed.
30-5-12	Pulse counts	70-144	288	None	Pulse slow and irregular while resting; fast and regular while standing.

DATE.	METHOD OF EXAMINATION.	Vs RATE	As RATE.	DRUGS.	REMARKS.
1-6-12	Polygraphic	72-144	288	None	Chiefly 4:1 but often irregular. Fast standing. No change of auricular rate with posture.
3-6-12	Pulse counts	80-140		None	Pulse slow and irregular while resting. Fast and regular while standing.
4-6-12	Pulse counts	70-90	Rapid	Tinct. Stroph. m. xl	Pulse slow and irregular while resting. Fast and regular while standing.
5-6-12	Electrocardiographic	146	292	1 drachm	Slower after lying some while.
6-6-12	Electrocardiographic	142	284	1 drachm	2:1, slower and irregular on lying.
7-6-12	Electrocardiographic	144	288	1 drachm	2:1, slower and irregular on lying.
8-6-12	Electrocardiographic	80-142	284	m. xl	2:1 chiefly. Occasional periods of 4:1.
9-6-12				1 drachm	
10-6-12	Electrocardiographic	75-142	284	1 drachm	2:1 with frequent periods of 4:1 and 5:1.
11-6-12	Electrocardiographic	80-145	290	1 drachm	Chiefly 2:1. Periods of 4:1 and 5:1 frequent on lying or resting.
12-6-12	Electrocardiographic	72-144	288	1 drachm	Chiefly 2:1. Periods of 3:1, 4:1 and 5:1 on lying or resting.
13-6-12				1 drachm	
14-6-12	Electrocardiographic	80-139	278	1½ drachms	Chiefly 2:1. Periods of 4:1 while lying or resting.
15-6-12	Electrocardiographic	75-150	300	1½ drachms	Chiefly 2:1. Periods of 3:1 and 4:1 while resting.
16-6-12				1½ drachms	
17-6-12	Electrocardiographic	150	300	1½ drachms	Chiefly 2:1, irregular while resting.
18-6-12*	Electrocardiographic	144	288	Stroph. stopped. (Total dr. xv, m. xx)	Chiefly 2:1, irregular while resting.

*The treatment was abandoned on the 18th, as there seemed little or no prospect of obtaining fibrillation. Diarrhœa had been present for a number of days.

The patient's general condition had improved a good deal, chiefly it may be presumed as a result of rest and the slower ventricular action, whenever he was lying or quiet. During the whole of the treatment he rested in bed or upon a couch, getting up for a few hours each day.

13-9-12. The patient writes that his heart rate is still fast.

Two figures are published from this case. The first (Fig. 28) was taken when the patient first came under observation. It shows a regular auricular action at 324 per minute; the ventricle beating at half this frequency. The auricular complexes are clearly visible in all three leads. In lead *I* they are small, pointed, upwardly directed peaks. The *P-R* interval is of about .09 sec. duration. In leads *II* and *III* the auricular complexes are contiguous, together forming a wavy line. The point which represents the onset of auricular systole in these leads can be ascertained. The quick deflections (*P*) of lead *I*, must be held to mark the commencement of systole (for at the commencement of systole the potentials of right and left side are unbalanced). Taking the same *P-R* intervals for leads *II* and *III*, we arrive at the commencements of the upstrokes. *P*, therefore, is convex in each of these leads and starts in an upstroke. *T* is seen only in lead *I*.

Fig. 29 was taken from the same subject four months later while he was under digitalis. A higher grade of heart-block had developed as a result of the drug administration. The auricular rate is 308. The ventricular action is very irregular, consisting of a mixture of 2 : 1 and 5 : 1 ratios. The *P-R* intervals, which vary in length, will be referred to in more detail at a later stage.

Summary. In a man of 53 years, attacks of palpitation and rapid heart action had been present for 38 years. When first seen the rapid action had been persistent for three years. An auricular rate which varied between 264 and 324 was recorded and was persistent over a period of observation of five months. The ventricle responded to each second auricular contraction. Posture had no influence on the auricular rate; a continued course of digitalis appeared to reduce it somewhat. Under full doses of digitalis the ventricular rate was retarded, the pulse becoming extremely irregular. The digitalis was withdrawn before fibrillation of the auricles could be induced. Tincture of strophanthus was used and produced similar ventricular slowing. Pressure on the vagus (right and left) produced increased ventricular, but no auricular slowing, while the patient was under the influence of these drugs. The treatment with strophanthus had also to be abandoned. The general condition of the patient at the end of treatment seemed improved.

CASE 4. W. T., a traveller, aged 62, came under observation on May the 24th, 1912.

Past illnesses. When young he had scarlet fever and smallpox. Until the age of 35 his health remained good; he then had an attack of appendicitis. At the age of 40 he developed a hernia. At 42 he had inflammation of the lungs and a slight attack of typhoid fever. At 45 he developed a fistula and subsequently hæmorrhoids. He was operated upon twice for the rectal conditions and subsequently was not bothered by them.

History of cardiac condition. In the late months of the year 1911 he noticed a little shortness of breath on exertion. In the first week of January, 1912, he was laid up with a severe attack of "influenza"; the illness commenced with headache, prostration, cough and fever (100-101° F.); he was in bed for eight weeks and during that time his doctor noticed that his heart began to beat rapidly. The patient noticed nothing the matter with his heart until his attention was drawn

to it. He got up early in March and experienced feebleness and considerable shortness of breath upon exertion. He was easily exhausted and had sharp short pains localised to the left side of his chest. The pains were not influenced by exercise or by food. They came on at any time of the day or night, and often while he was sitting quietly. Usually they lasted but a few minutes. He also suffered from cramping pain and soreness of the left shoulder which usually disturbed him most when in bed; the shoulder pain was present while he was under treatment. Food was always taken well, though he had suffered from slight indigestion at times, and the bowels were generally constipated. The heart's action remained continually rapid.

Condition on May the 24th, 1912. A heavily built man with florid countenance, he had a trace of cyanosis. His breath was somewhat short and the slightest exertion enhanced this symptom, so that in walking up a gentle declivity he was obliged to halt every few hundred yards. The limits of cardiac dulness lay $2\frac{1}{2}$ and $6\frac{3}{4}$ inches to the right and left of the mid-sternal line, respectively. The heart sounds were natural and there were no murmurs. The heart's action was rapid. The arteries showed no signs of thickening; the systolic blood pressure was 140 mm. Hg. The urine was normal. Neither the liver nor the spleen could be felt. The teeth were carious.

The patient was examined on two occasions before observation became continuous. He was treated first with digitalis and secondly with strophanthus. The observations are embodied in the following tables.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
19-3-12	Electrocardiographic	138	276	None	2 : 1 heart-block.
22-5-12	Electrocardiographic	138	276	None	2 : 1 heart-block.
24-5-12	Polygraphic	131	262	Tinct. Digit. m. x	
25-5-12	Polygraphic	136	272	m. xxx	2 : 1 heart-block.
26-5-12	Electrocardiographic	136	272	m. xxx	2 : 1 heart-block.
27-5-12				m. xxx	
28-5-12	Electrocardiographic	135	270	m. xlv	2 : 1 heart-block.
29-5-12	Electrocardiographic	138	276	m. xlv	2 : 1 heart-block.
30-5-12	Electrocardiographic	114-137	274	m. xlv	2 : 1 with occasional 4 : 1 periods. A little pain in left chest. Pressure on right vagus slowed ventricle but not auricle.
31-5-12	Electrocardiographic	94	278	m. lx (Total dr. iv, m. lv)	2 : 1 and 4 : 1 mixed. Very irregular.

On the 31st the patient commenced to vomit. The digitalis was omitted and, after a few days rest, strophanthus was given.

1-6-12	Polygraphic	100-130	260	None	2 : 1 with 4 : 1 periods. In bed, sickness stopped.
3-6-12	Polygraphic	90-132	264	None	Periods of 2 : 1, also 3 : 1, 4 : 1 and 5 : 1. No sickness or nausea.
4-6-12	Electrocardiographic	110-134	268	Tinct. Stroph. m. xx	Vagal pressure (right) produced ventricular but not auricular slowing. 2 : 1 and 4 : 1 mixed.
5-6-12	Electrocardiographic	136	272	m. lx	2 : 1 heart-block.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
6-6-12	Electrocardiographic	100-135	270	Tinct. Stroph. m. lx	Slight diarrhœa and giddiness. 2:1 heart-block. Some 4:1 periods.
7-6-12	Electrocardiographic	125-135	270	m. lx	Chiefly 2:1. Some 4:1 periods lying.
8-6-12	Pulse counts	80-100		m. lx	Very irregular; in bed.
9-6-12				m. xl	
10-6-12	Polygraphic	130	260	m. lx	2:1 occasional periods of 4:1. Had just been dressing himself. Pressure on either vagus caused slowing.
11-6-12	Electrocardiographic	115-136	272	m. lxx	Chiefly 2:1, occasional periods of 4:1.
12-6-12	Electrocardiographic	100-133	266	m. lxxv	2:1 with frequent 4:1 periods, especially while resting.
13-6-12	Electrocardiographic	105-135	270	m. l	2:1 with frequent 4:1 periods, especially while resting.
14-6-12	Electrocardiographic	95-135	270	m. xxv	2:1 with frequent 4:1 periods, especially while resting.
15-6-12	Electrocardiographic	98-134	268	m. l	2:1 with frequent 3:1 4:1 and 5:1 periods.
16-6-12				m. lxxv	
17-6-12	Electrocardiograms	90	275	m. lxxv	Very irregular, 2:1, 3:1, 4:1 and 5:1 periods mixed.
18-6-12	Electrocardiograms	95	273	m. l	Very irregular, 2:1, 3:1 and 4:1 periods mixed.
19-6-12	Electrocardiograms	92	276	m. lv	Very irregular, 2:1, 3:1 and 4:1 periods mixed.
20-6-12	Electrocardiograms	80	270	m. xxx Stroph. stopped. (Total dr. xv, m. xv)	Very irregular. 3:1 and 4:1 mixed. Diarrhœa has been present since the 6th.
23-6-12	Polygraphic	132	264	None	Chiefly 2:1. Occasional 3:1 and 4:1 periods. Diarrhœa stopped. Has improved in general condition. Vagal pressure slows the ventricle.
23-6-12. On this date the treatment was abandoned as there seemed no prospect of obtaining fibrillation.					
19-7-12	Polygraphic	133	266	None	Pressure on right vagus produced long pauses.
14-8-12	Electrocardiographic	136	272	None	Health unaltered.
11-9-12	Electrocardiographic	135	270	None	Health unaltered.

Observations were made upon the heart rate as affected by posture and exercise. On the 24th of May, 1912, curves were taken from the brachial artery in the standing and lying postures ; the pulse was regular throughout, the ventricle beating at half the auricular rate.

Lying.	Standing.
130	130
130	132
130	—

Similar observations were made upon June the 23rd, 1912.

Lying.	Standing.
129	134
132	134
130	133

Similar observations were made on July the 19th, 1912.

Lying.	Standing.
133	132
132	132
130	133

On June the 23rd, 1912, two continuous curves were taken immediately after exercise.

Rate before exercise (standing) 134.

Rate each half minute after exercise (standing) 134, 134, 134.

Rate each half minute after exercise (standing) 134, 134, 134, 134.

Another curve of the same sort was taken on July the 19th, 1912.

Rate before exercise (standing) 135.

Rate each half minute after exercise (standing) 135, 134, 136, 137, 137, 137, 136, 136.

The electrocardiograms from this case are very similar to those of *CASE 3*. The two published curves (Fig. 30 and 31) were taken within two days of each other, the second (Fig. 31) shows the first reaction of the heart to digitalis. In Fig. 30 the ratio is as 2 : 1, the auricular rate being 276 per minute. *P* is not clear in lead *I*, but in leads *II* and *III* the continuous line of auricular complexes has a form almost identical with that seen in Fig. 19 and 20 ; but there is a difference. In Fig. 20 similar phases of the wavy lines correspond in leads *II* and *III* ; while in Fig. 30, the upstroke of each wave in lead *II* corresponds to a point a little to the right of the upstroke in lead *III*. *T* appears in lead *I* only.

The irregular heart action of Fig. 31 consists of responses to each second, third or fourth auricular systole ; there is as usual a variation in the length of the *P-R* interval, according as it falls after a long or short ventricular cycle.

Summary. A man of 62 years developed a continuously rapid heart action during a severe attack of “influenza.” The auricular rate was 270, the ventricular rate 135 per minute. After the auricular “flutter” had been present for five months, he was treated first with digitalis and secondly with strophanthus. These drugs slowed the ventricle but had no influence upon the auricle. Vagal (right and left) pressure, while he was under the influence of both these drugs caused further ventricular slowing without affecting the rate of the auricle. Posture influenced the rate of the auricle but a few beats per minute ; exercise had no effect. The treatment with both digitalis and strophanthus was abandoned ; fibrillation could not be obtained. There was slight but definite improvement in the condition, presumably as the result of the rest and drug administration.

CASE 5.* W. G., a French polisher, 60 years of age.

Past history. The patient had gonorrhœa at the age of 17. At 23 he was laid up with some obscure chest ailment.

History of cardiac condition. There had been periodic attacks of breathlessness of a distressing and exhausting character for three and a half months.

Condition, November the 9th, 1911. Orthopnoea and slight cyanosis were present. The pulse was constantly rapid and regular. The apex beat lay in the fifth and sixth spaces; the limits of heart dulness were $1\frac{1}{4}$ and 6 inches to right and left of the mid-sternal line. A systolic murmur was heard at the apex. The liver was enlarged; the urine was normal. The blood pressure varied between 116 and 120 mm. Hg.

The mechanisms and the rates of heart beat are summarised in the following table which shows the course of the illness under treatment.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
19-11-11 to 25-11-11	Pulse counts	145-150		None	In all probability 2:1 heart-block throughout.
25-11-11	Pulse count	150		Digit. infus. dr. xii	2:1 heart-block. The rate was not changed by posture.
26-11-11	Electrocardiographic	146.5	293	dr. xii	2:1 heart-block. Occasional premature beats from the ventricle.
28-11-11	Electrocardiographic	150	300	dr. xii	2:1 heart-block.
1-12-11	Electrocardiographic	74.8- 149.5	299	dr. xii	2:1 heart-block with occasional 4:1 periods.
2-12-11	Electrocardiographic	72-100	288	dr. xii	4:1 heart-block with occasional 2:1 periods.
3-12-11	Polygraphic	73	292	dr. xii	4:1 heart-block.
4-12-11	Electrocardiographic	72	288	dr. xii	4:1 heart-block. Pressure on either carotid sheath gave further slowing of the ventricle but none of the auricle.

The condition remained unaltered until December the 11th, 1911.

11-12-11	Electrocardiographic	44	0	Digit. infus. dr. xii	Gross irregularity. Auricular fibrillation.
12-12-11	Polygraphic	46	0	dr. xii (Total xxvii oz.)	Gross irregularity. Auricular fibrillation. Digitalis stopped.
13-12-11	Polygraphic	45	0	None	Auricular fibrillation.
14-12-11	Polygraphic	56	0	None	Auricular fibrillation
18-12-11	Polygraphic	53	0	None	Auricular fibrillation.
20-12-11	Polygraphic	62	0	None	Auricular fibrillation.

* This case has been fully reported in *Heart*, 1912, III, 285. I repeat the chief facts of the case in the form of a summary, and enter the case in this section of the communication because I am able to add to the first report.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
4-1-12	Electrocardiographic	63	63	None	Normal rhythm. Occasional premature auricular contractions.
6-1-12	Electrocardiographic	63	63	None	Normal rhythm. Occasional premature auricular contractions.

8-1-12. Discharged from hospital, much improved ; colour good, no liver enlargement, breathing free and easy.

20-1-12	Electrocardiographic	82	82	None	Normal rhythm. Pressure on neither carotid sheath had any effect.
24-2-12	Electrocardiographic	132	264	None	Has been feeling bad again for a week. 2 : 1 heart-block present.
27-4-12	Electrocardiographic	68-136	272	?	Chiefly 2 : 1 heart-block. A good many 4 : 1 periods.

Re-admitted to hospital 18-5-12, complaining of pain in the chest, shortness of breath, palpitation and swelling of the abdomen and feet. The pulse was continuously rapid. Slight cyanosis, dyspnœa, liver enlargement, ascites and dropsy of the feet were present.

18-5-12	Pulse counts	145		Inf. dig. dr. xii daily	
22-5-12	Electrocardiographic	69-138	276	dr. xii	Chiefly 4 : 1 heart-block, some 2 : 1 periods.
29-5-12	Electrocardiographic	72-144	288	dr. xii	Chiefly 4 : 1 heart-block, 2 : 1 periods after effort.
1-6-12	Electrocardiographic	64-90	256	dr. xii	Chiefly 4 : 1 heart-block, some 2 : 1 and 3 : 1 periods. Pressure on either vagus produced further slowing.
5-6-12	Pulse count	67		dr. xii	The pain has gone, and all the signs of liver enlargement. Ascites and dropsy have disappeared. Pulse regular.
6-6-12	Pulse count	60		dr. xviii daily	Pulse regular.
9-6-12	Polygraphic	62	248	dr. xviii daily.	4 : 1 heart-block.
15-6-12	Polygraphic	58-64	256	dr. xviii daily	Chiefly 4 : 1 heart-block, some 8 : 1 periods ; pressure on either vagus produced further slowing.
20-6-12	Polygraphic	54-62	248	Digit. stopped. (Total lxii oz.)	Chiefly 4 : 1 heart-block, some 6 : 1 and 8 : 1 periods.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
24-1-12	Pulse counts	80-110		None	Very irregular.
27-6-12	Electrocardiographic	62.5-125	250	None	General condition very much improved. Pressure on vagi still induces ventricular slowing. While the patient is lying the ratio is 4 : 1 ; after exercise it is 2 : 1 or irregular.
27-6-12 Discharged from hospital.					
6-7-12	Electrocardiographic	95	0	None	Auricular fibrillation.
3-8-12	Electrocardiographic	85-120	85-120	None	Normal rhythm. Pressure on right and left vagi produced great slowing of whole heart, with slight prolongation of P-R interval.
14-9-12	Electrocardiographic	65	65	None	Normal rhythm. Occasional premature auricular contractions.

Observations were made as to the effect of posture on the auricular rate (calculated from the regular ventricular rhythm) in this case on a number of occasions. The effect was always negative or inappreciable. An example of readings may be given :—

Lying.	Standing.
249	250
249	248
251	245
249	246

Exercise, similarly, was without appreciable result. Before exercise the auricular rate was 246, at half-minute intervals, directly after exercise, the readings were 244, 246, 245 and 252 ; after further exercise, they were 256, 254, 254 over intervals of one minute.

Control curves were taken on September the 3rd, while the mechanism was normal. The rate (auricle and ventricle) before exercise was 75, after exercise 110 and later 80. Postural effects were also observed.

Lying.	Standing.
84	111
79	110
82	112
84	111

The electrocardiograms of this case were fully illustrated in my former paper. The *P* summits of the periods of flutter were clear in all leads ; and those of lead *I* were small, pointed and upright summits. In leads *II* and *III* the lines of auricular waves were contiguous, the separate summits being convex ; the rate was about 300 per minute.

Curves showing, chronologically, mixed 2 : 1 and 4 : 1 periods, regular 4 : 1 block ; auricular fibrillation ; and lastly, the normal rhythm were published. The changes occurred as a result of digitalis administration. The auricular oscillations were more conspicuous during the period of flutter than during the period of fibrillation. The *P* summits of the normal rhythm were quite unlike those of the period of flutter. The shape of the initial

ventricular deflections remained unchanged throughout. *T* was scarcely visible in any lead.

Summary. A man of 60 years developed a continuously rapid heart action. The auricular rate was 300, the ventricular 150. After the "flutter" had been present for some while, he was treated with digitalis. This drug slowed the ventricle to a fourth of the auricular rate. During this stage, pressure on either vagus caused further slowing of the ventricle but had no effect upon the auricular rate. Eventually fibrillation appeared and, the digitalis being stopped, the normal rhythm reappeared. Some seven weeks later the flutter came again and digitalis, though it produced conspicuous slowing, seemed to have no influence upon the auricles. Eventually he was discharged and a week later fibrillation was found; this subsided and the normal rhythm was again restored. The flutter rate was influenced by posture or exertion to no appreciable extent; the normal rhythm, when present, was influenced by both, and vagal pressure (right and left) gave slowing, during this stage.

CASE 6. F. T., an actor of 47 years.

On June the 18th, 1912, Dr. Jordan, of Guy's Hospital, asked me to examine a case of tachycardia. The patient had been to Dr. Jordan in the morning for an examination of the alimentary canal. Looking at the heart upon the fluorescent screen, Dr. Jordan could see an extremely rapid undulation of the heart margin; the systolic displacement of the ventricular border was almost absent, a tremulous movement of the left outline was alone visible. At the time, the pulse was practically imperceptible.

In the evening I saw the patient at Dr. Jordan's rooms. He seemed in no distress, the breathing was free. The ventricular action was quite regular, the rate being about 160 per minute. The pulse was also regular and fairly full. The systolic displacement of the left heart border was clearly visible on the fluorescent screen and amounted to $\frac{1}{4}$ or $\frac{1}{2}$ an inch. The tug of the ventricle upon the right auricle was clear, but no intrinsic beating could be seen in this auricle. On the other side, in the position of the left appendix, there was a movement of much greater rapidity. The arch of the aorta showed slight senile dilatation. The heart limits to percussion and as seen upon the screen were a little wide towards the left side. There were no murmurs. The arteries seemed soft and the tension moderate. The same evening electrocardiograms were taken. The patient gave a history of attacks of palpitation coming on suddenly and being accompanied by rapid heart action ever since he was a young man. The attacks had left him exhausted and unfit for exertion. They were brought on by exercise or emotion; he had had them while on the stage, but had always managed to complete his evening's work, though often only with considerable effort. For years he had suffered from attacks of acute gout in the feet and wrists, the joints becoming inflamed, swollen and tender. The condition moved from joint to joint with suddenness. There were no tophi in the ears, but chronic changes were found in the metatarsophalangeal joints of the great toes and in the wrists. The patient refused treatment.

The electrocardiograms showed an extremely rapid auricular action, the rate being 330. The ventricle was beating at half this rate. The electrocardiograms are shown in Fig. 32. *T* is present in leads *I* and *II* but not in lead *III*. The auricular summits in lead *I* are small and inconspicuous, though visible. In leads *II* and *III* they are of the usual form, and are contiguous. (Compare Fig. 29 and 31.) It is probable, but not certain, that each auricular cycle commences with an upstroke.

Summary. A man of 47 years had suffered since he was a young man from attacks of rapid heart action. He suffered also from gout. He was

seen on one occasion only; the auricles were found to be beating at 330 per minute and the ventricles at 165 per minute. It is probable that on occasions the ventricular action responded more rapidly; in other words that the grade of 2:1 heart-block gave place to one in which the responses were successive.

CASE 7. A. P., an elderly gentleman, was examined electrocardiographically only and I have notes neither of his symptoms nor of the size of the heart or its auscultatory signs. The electrocardiograms are shown in Fig. 33. The heart's action is quite regular. The auricular rate is 228, the ventricular rate 114. 2:1 heart-block is present. The auricular summits are most distinct in lead *II*, but are also clearly visible in leads *I* and *III*. In all leads the summits are upright. The curves differ from the others of this series in that the *P* summits are not contiguous; distinct intervals are present between the termination of the downstrokes and the commencement of upstrokes. This fact is probably accounted for by the slow rate of auricular action as compared with that found in other cases. The heart action was a persistent one, having been present for some months.

CASE 8. W., a clergyman of 52 years, was seen on a single occasion and for electrocardiographic examination only.

I have but brief notes of his history. For some while he had suffered from breathlessness and a sense of weakness and exhaustion whenever he exerted himself. He was unable to walk fast, though with effort he could manage to keep going for a good many miles. He had been treated for albuminurea some years before and was reported cured. There was some cardiac enlargement to the left, but no murmurs; the heart action was very irregular.

The electrocardiograms showed that the auricle was beating regularly at 260 per minute. The ventricular rate varied between 90 and 130. As a rule the ventricle responded to each alternate auricular contraction; 3:1 and 4:1 periods occurred from time to time. The ventricular complexes were very similar to those seen in the other patients. In leads *II* and *III* the auricular complexes were contiguous and were of the customary form, forming as a whole a zig-zag line, composed of short steep upward deflections and longer and more inclined downward deflections. The type of curve is shown in Fig. 29. The summit *T* could not be found in any lead.

Previously reported cases.

CASE 9. (Reported by Hertz and Goodhart.¹) A woman of 39 years.

There was a history of scarlet fever at the age of 11, after which she suffered from palpitation; for this she was treated eight years later and became perfectly well. A few months before she was examined she became breathless and developed lividity. The heart beat irregularly at this time.

The patient had an organic hemiplegia. There was but little cardiac enlargement, a systolic apical murmur was present, the rate of beat was slow. At times a presystolic or early diastolic murmur was heard. The ventricular beats were coupled. On digitalis the ventricular rate fell and reached 44 beats per minute.

Polygraphic curves showed an auricular action varying between 216 and 234 beats per minute. Atropine and exercise had no effect on the auricular rate but increased the ventricular rate. A high grade of partial heart-block is shown in the single curve illustrating the case. She was under observation for nearly a year and the auricular action was constantly fast over this period.

CASE 10. (Reported by Jolly and Ritchie,² *CASE 1*). A man of 61 years.

The ventricular action was always slow, varying between 30 and 63 beats per minute, the usual rate being in the neighbourhood of 33 beats per minute. He had suffered from complete heart-block, the auricle and ventricle beating independently at the usual rates found in such cases, and in addition, attacks of great acceleration of the auricle were observed, the last of which became permanent and was watched for two years. The auricular rate, while it was accelerated, varied between 234 and 300 per minute. The only symptoms mentioned were those of Adams-Stokes' syndrome. Electrocardiograms and polygraphic curves are given; they show complete block and an extremely fast auricular action. The type of auricular curve was almost identical with that shown in Fig. 29 of this paper. Digitalis apparently was not used. Atropine was given and had no influence upon the auricular and ventricular rates.

CASE 11. (Reported by Rihl,⁹ *CASE 1*.) A cabinet maker of 55 years.

The patient was said to have suffered from typhoid at 18 and pneumonia a year later. For many years afterwards he had painful swellings of single joints of a few days duration. He gave a history of four months' palpitation and breathlessness, which were especially prominent after exertion.

The apex beat was in the fifth and sixth spaces. The left border of dulness lay a finger's breadth outside the medio-clavicular line; the right border at the left edge of the sternum. The blood pressure was 86-124 mm. Hg. There were no murmurs. Some of the arteries were tortuous. The progress of the case is summarised in the following table.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
2-6-10	Polygraphic	143-150	Probably 246-300	None	
5-6-10				Digalen m. xvii	
6-6-10	?	150	Probably 300	m. l	
7-6-10	Polygraphic	100	Probably 290	m. xxxiv	Heart action irregular. 2 : 1 and 4 : 1 periods.
8-6-10	Polygraphic	About 85	Probably 304	m. xxxiv	Heart action irregular. 4 : 1 with 2 : 1 and 3 : 1 periods.

DATE.	METHOD OF EXAMINATION.	Vs RATE.	As RATE.	DRUGS.	REMARKS.
9-6-10				m. xxxiv	
10-6-10				m. xxxiv	
11-6-10	Polygraphic	About 74	About 296	m. xxxiv (.0006 gr. Atropine)	Atropine injection temporarily restored original condition. Rates 140 and 280.
12-6-10				m. xxxiv	
13-6-10	?	76		m. xxxiv	Irregularities infrequent. Probably chiefly 4 : 1.
18-6-10					Systolic murmur audible at apex.
23-6-10	?	Approx. 100			Very irregular again.
24-6-10	Polygraphic	Approx. 95	Approx. 288	Atropine .001 gr. Digalen m. xvii	Atropine restored original 2 : 1 rhythm. (Rates 141 and 282).
25-6-10				m. l	
26-6-10	?	Approx. 75		m. l	Irregularity infrequent.

Rihl gives electrocardiograms and polygraphic curves which are characteristic of the condition. The auricular curves are almost identical with those seen in Fig. 29 of this paper. The *P-R* intervals are similar. The auricular representative commences with an upstroke in leads *II* and *III*, and are not, as Rihl states, inverted. *T* is clearly visible in lead *I* only.

To sum up, the case was one in which tachycardia had probably been present for four months ; the ventricular rate was 143-150 ; the auricular rate was 286-300. Digalen reduced the ventricular but not the auricular rate and led to irregularity and slowing of the pulse. Atropine abolished this digitalis effect. Pressure on the vagus increased it but did not alter the auricular rate.

CASE 12. (Rihl,⁹ *CASE 2.*) A widow of 32 years and a waitress.

At 24 years she had typhoid and pneumonia. Two years later her knees were swollen. Palpitation and dyspnœa, especially after strenuous exercise, had been present for a year ; for four weeks the feet had been swollen. The apex beat lay in the sixth interspace in the anterior axillary line. No extension of dulness could be found to the right of the sternum ; the left border lay beyond the mid-clavicular line. A conspicuous thrill was felt at the apex and two murmurs were heard. The urine flow was not free. The blood pressure lay between 92 and 112 mm. Hg.

Polygraphic curves showed an auricular rate of 206-222. The ventricular rate varied, being sometimes half the auricular, the pulse then being regular, or sometimes less, the pulse being then usually irregular. Digitalis gave

rise to ventricular slowing and irregularity. Vagal pressure increased the block, but whether the heart was under digitalis at the time is uncertain. The patient was under observation from time to time for a period of 18 months and the fast auricular action seems to have been present during the whole of this period.

CASE 13. (Rihl,⁹ CASE 3.) A labourer of 72 years.

A year before coming under observation he suddenly became afflicted with palpitation and developed cough and swelling of the legs. The symptoms disappeared after a long stay in hospital. For three weeks before admission he had had the same symptoms.

The apex beat lay in the sixth space. The heart dulness was considerably increased to the left. There was no murmur. The blood pressure lay between 145 and 200 mm. Hg.

The case was examined by means of polygraph curves. The ventricular rate was half the auricular, the latter being between 200 and 214 per minute. The ventricular responses were sometimes slower and irregular. Digitalis increased the grade of heart-block. So also did vagus compression, but it is not clear whether the heart was under the influence of digitalis at the time. On three occasions curves were taken which showed what seems to have been fibrillation of the auricles. The change apparently did not result from digitalis administration. The ventricular rate was then 120 or less.

Three cases in which the condition was not recognised.

CASE 14. (Reported by Mackenzie,⁸ CASE 37; the curves were fully interpreted by the writer at a later date.⁷) An army official, aged 47, who gave no history of past illnesses.

History of heart condition. For eight years he had suffered from attacks of palpitation. For three years they were not accompanied by much distress; after that time he had a number of attacks and had to rest. After an interval of several years of freedom from them they returned, and when seen he had been suffering from attacks of much greater severity, accompanied as they were by great exhaustion and prostration.

Condition on May the 3rd, 1910. Cyanosis and orthopnoea were present. The pulse was soft and regular, the rate being 140; the blood pressure was 140 mm. Hg. The heart's dulness lay 0 and 4½ inches to the right and left of the mid-sternal line. There were no murmurs.

Progress. For five weeks the ventricular rate was maintained, lying usually between 130 and 150, but rising on occasion to a rate of 290 and 300. On July the 22nd, or about eleven weeks after admission, the pulse remaining rapid, he was given tincture of digitalis in doses of 20 minims three times a day. Five days later the pulse rate dropped to 55 and the ventricular action became very irregular, though there were still occasional rapid and regular paroxysms. On the 28th, periods of normal rhythm and irregular heart action succeeded each other. On this day the digitalis was stopped but was resumed on the 30th and continued till August the 7th in half doses. Irregular action was present from the 2nd till the 10th of August. From August the 17th to the 24th, normal rhythm, regular tachycardia and irregular action occurred from time to time. After the 24th the regular tachycardia became more frequent and digitalis was again given on September the 4th in doses of 5 minims three times a day and later 10 minims a day. Under this régime the tachycardia gradually disappeared; after the 29th it was not seen.

Summary. It may be said that the patient suffered from attacks of a curious form of regular tachycardia, which gave place under digitalis to a slow irregular action, and that finally the normal rhythm was restored, the auricle and ventricle beating at the same rates. Electrocardiograms taken during the periods of regular tachycardia showed a ventricular rate of 160

per minute and an auricular action of 320 per minute. The auricular curves were very similar to those shown in Fig. 29 of the present paper. Each commenced with an upstroke. *T* was present in lead *I* only. Electrocardiograms taken during the normal periods showed similar ventricular electric complexes, but the auricular complexes were very different from those of the period of flutter. The polygraphic curves published of the slow irregular action indicate the probable appearance of auricular fibrillation from time to time.

CASE 15. (Reported by Turnbull.¹¹) A retired medical practitioner aged 74.

The patient gave a history of no previous illness; he had been a healthy and athletic man; he stated that he awoke one morning in January, 1910, feeling ill. His doctor found a rapid ventricular action, and advised rest, but the patient continued his usual occupations, until increasing breathlessness compelled him to lie up. The regular and rapid ventricular action continued for four months. He was treated with digitalis, 20 minims being given three times a day. After seven days treatment a change was noticed in the heart action; it became slow and irregular (probably as a result of increased heart-block). In the venous curves, taken at this time, certain additional waves the meaning of which was obscure at that time (Fig. 1 and 2 of the original paper) were observed. Later the regular tachycardia was re-established. Irregular action again appeared and strips of curve (Fig. 7 of the original paper) showing fibrillation were obtained. Eventually the normal rhythm was restored. Electrocardiograms were published showing the action during the regular and fast periods and also of the restored normal rhythm. I may add that I have recently taken curves from the same subject (June, 1912) and that the normal rhythm has been maintained.

If Turnbull's paper is referred to and the tracings are compared with those of the present communication, it will be obvious that the condition dealt with was one of auricular flutter. In the original paper, I interpret Fig. 1 *a* and *b*, 3 and 4 as showing periods of regular ventricular acceleration with an auricular rate which is exactly double. All the venous waves in Fig. 3 and 4 are auricular. The irregular periods of Fig. 1 *a* and *b*, and Fig. 2 and 6, are I think undoubted examples of irregular response to an auricle whose regular and accelerated beating is maintained. This new interpretation entirely accounts for the additional waves in the venous curves which were so puzzling to Turnbull. Fig. 7 is almost certainly an example of auricular fibrillation.

The electrocardiograms which accompanied the report could not be satisfactorily interpreted at the time, but I think that now the interpretation is clear. I republish the figure as Fig. 34 of this paper and have rearranged the figure and have marked the outline of the auricular representatives on the curve. They are of similar forms to those shown in the other cases now reported, though much more difficult to distinguish. They probably commence in upstrokes as in the other cases.

The upstroke of the auricular beat which commences during a *QRS* phase, almost coincides with the upstroke of *S* in leads *II* and *III*, as now indicated by the dotted lines. The *P* summits of the normal rhythm were quite dissimilar. During the flutter period, *T* is present in lead *I*, but hardly discernible in leads *II* and *III*.

We may summarise Turnbull's case as follows :—The patient had suffered from a condition in which the ventricle beat at 140-150 per minute, the auricle at 280-300 per minute, for four months. As a result of digitalis administration a further and irregular grade of heart-block was established; subsequently auricular fibrillation appeared, and eventually the normal rhythm was restored and has been maintained for two years.

*CASE 16.** (Reported by the writer in conjunction with H. G. Schleiter,⁵ *CASE 1.*) A cabinet maker of 28 years, came under observation on a number of separate occasions.

Past illnesses. There had been no previous illness, except measles as a child and occasional colds.

History of cardiac condition (November the 11th, 1910). The first attack was in 1908, and from that time on he was subject to them every few months. The attacks were accompanied by palpitation, breathlessness, fainting, fatigue, salivation, sweating, sickness and great prostration.

Condition between attacks. A well-built man of healthy aspect. The cardiac limits were normal to percussion: there was a short systolic thrill and murmur, otherwise the heart sounds were normal. The arteries showed a little thickening. The blood pressure lay between 70 and 120 mm. Hg. There were no other physical signs.

The attacks, a number of which were closely observed, consisted almost always of paroxysms of auricular fibrillation. On one occasion a paroxysm of regular tachycardia of twelve hours duration was observed. A similar mechanism was observed on a second occasion at the end of a paroxysm which started as auricular fibrillation and lasted for three and a half days. Electrocardiograms of the three states, *i.e.* normal rhythm, regular tachycardia, and auricular fibrillation were published in the original account of this case. The interpretation of the curves showing regular tachycardia have now to be revised in the light of more recent observations. It has become apparent that though the interpretation is correct in so far as it has proceeded, yet an additional auricular systole has passed unnoticed in each ventricular cycle. I republish the curve as Fig. 35 in the present paper. The auricular action is especially well seen in the third lead. As a whole the auricular curves, which have been dotted, are very similar to those of most of the other cases of the series. *P* probably commences in an upstroke. The *P* summits in electrocardiograms from the normal periods were quite dissimilar. *T* was visible in lead *I*, but only just seen in lead *II* and absent in lead *III*. The ventricular rate is approximately 140, the auricular rate is approximately 280. This case is especially noteworthy because the “flutter” of the auricle was transient; because the auricular fibrillation passed into “flutter” and because it ended spontaneously. The case also illustrates the difficulty of recognising the mechanism, when the ventricle responds to each second auricular beat; the interpretation was impossible until other cases showing 4:1 periods were published.

* Since these pages were written I have seen a report by Josué and Chevallier (Bull. c. Mém. d. l. Soc. méd. d. Hôp., Dec, 1911. It was probably of the same nature as those here described.

The arterial curves.

A full description of the arterial curves which accompany auricular flutter is desirable; close examination of them often enables the diagnosis of the condition, and in all cases a great deal may be learnt from them.

I base the statements in this section of the paper upon a detailed analysis of a very large collection of curves, and use a few selected curves for illustration of the chief facts. The correctness of the analysis has been substantiated by electrocardiograms taken from the same case and usually upon the same days; on a number of occasions simultaneous arterial and electrocardiographic curves have been taken.

To appreciate fully the form and spacing of the arterial beats it is necessary that certain facts should be borne in mind.

1. High rates of ventricular action are often accompanied, where there is degeneration of the myocardium, by *pulsus alternans*. Alternation is evident in the arterial curves of auricular flutter and confuses the analysis.

2. The strength of arterial beats is materially influenced by the pauses which precede them. This is a statement which requires qualification. When we deal with cases of partial heart-block in which the auricular rates are normal and the pulse is irregular, it is customary to find that the arterial beats are of equal heights. All the pauses are of sufficient length to allow the ventricular contractions to be of maximal efficiency. It so happens that when we study the arterial curves of ordinary heart-block, little or no variation in the height of pulse beats is perceived. The arterial curves of auricular flutter, on the other hand, conform to the general rule, when the responses are rapid. Pulse beats which follow 2:1 periods are almost always weak. They resemble extrasystoles. We might modify our original formula, and embody it in a statement which is generally and uniformly applicable. In any given case, pauses of less than a given interval of time are followed by pulse beats which are relatively weak, and the weakness of the beats is evident, be the cause of the shortened pauses what it may.

3. The heart-block in auricular flutter is accompanied, as heart-block often is, by considerable variation of the *As-Vs* intervals; whereby the expected relation between the lengths of long and short cycles is modified. A long cycle is succeeded by a shortened conduction interval; a short cycle by a lengthened conduction interval. The beat, which is preceded by a short pause, is small, as I have said; therefore, the small beats or those which are least effective,* are the ones before which a long *As-Vs* interval may be conjectured.

* In speaking of less effective ventricular contractions I wish to avoid the controversy as to whether the arterial beat is weak because the contractility of the ventricle is smaller, or whether, on account of the shorter pause, there is lessened ventricular filling. I speak of a less effective ventricular beat simply in the sense that it has less effect on the arterial pressure.

4. The arterial pulse beats which are weak often have a relatively long presphygmic interval; they correspond to ventricular contractions which, as I have said, are already delayed. The delay in the ventricle is exaggerated in pulse curves and is consequently considerable.*

In the following paragraphs I shall refer again and more explicitly to these factors in describing certain forms of pulse irregularity.

In auricular flutter the pulse rhythm is often perfectly regular for long periods. This occurs, as in Jolly and Ritchie's case, when complete heart-block is present. The pulse is then slow. It also occurs, and far more commonly, when there is a uniform grade of partial heart-block. If the ventricle responds to each fourth auricular contraction, and the auricular rate is from 240 to 300 per minute, then the ventricle is beating regularly and has a rate of from 60 to 75 per minute (see Fig. 11 and 12). When a patient who is in this state is examined by ordinary methods, the true condition of the heart will always pass unnoticed. But the condition is an unstable one, and it may be suspected or recognised, because with exercise the pulse becomes either very irregular, or because it shows exact doubling of rate. The irregularity or doubling is due to a decrease of the block. The pulse becomes irregular when the 4 : 1 periods are mixed with 2 : 1 periods. Doubling occurs when a 4 : 1 ratio gives place to a 2 : 1 ratio. If the ventricle beats in response to each second auricular contraction, it beats regularly and its rate is from 120 to 150 (see Fig. 8 and 9). When the rate is so fast it often happens that alternation is present, and this is especially the case when a 2 : 1 rhythm† follows a long pulse pause (Fig. 6).

The most confusing curves are those in which the responses to auricular systoles occur *irregularly*. We may commence by considering relatively simple examples. Fig. 3 shows a period of 4 : 1 heart-block, interrupted by a single period in which there is a 2 : 1 ratio. I have marked with an asterisk the beat which occurs too early. The cycle which precedes it corresponds to two auricular systoles, that which succeeds it to four. Yet the latter is shorter than the regular 4 : 1 periods; and this helps to distinguish the beat from a true premature contraction or extrasystole. The disturbance is known to result from heart-block because the bracketed stretches of curve are exactly equal. The two shorter cycles are exactly equal to $1\frac{1}{2}$ cycles of the regular rhythm; each of the bracketed stretches corresponds to six auricular systoles. The 2 : 1 period of the disturbed stretch is shorter than might be expected, and the 4 : 1 period of the same stretch is longer than might be expected, because the weak pulse beat is delayed; and it is delayed because the corresponding *As-Vs* interval is relatively long.

* Rihl has also referred to these changes at intervals. They are specially well seen in Fig. 36 and 37.

† In the accompanying figures, the actual ratio of the auricular to ventricular rate is marked above the individual pulse beats.

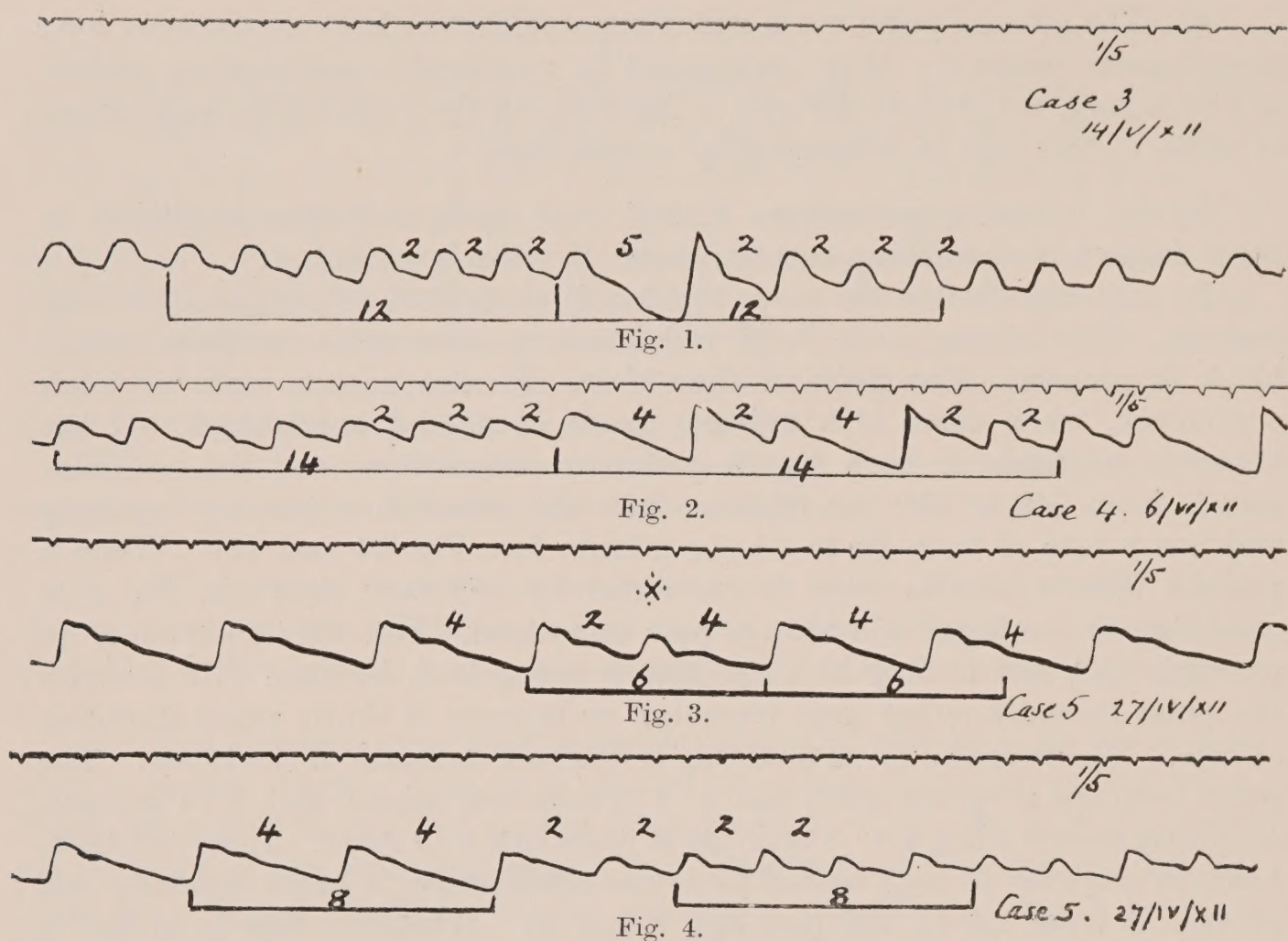


Fig. 1-4. Four arterial curves from cases of auricular flutter. They are published to show the methods adopted in analyses of these curves. The number of auricular cycles to the ventricular cycle is marked above the respective pulse beat. The bracketed portions of any single curve are of equal length; the number of auricular systoles corresponding to the beats included in a bracket is marked on the bracket.

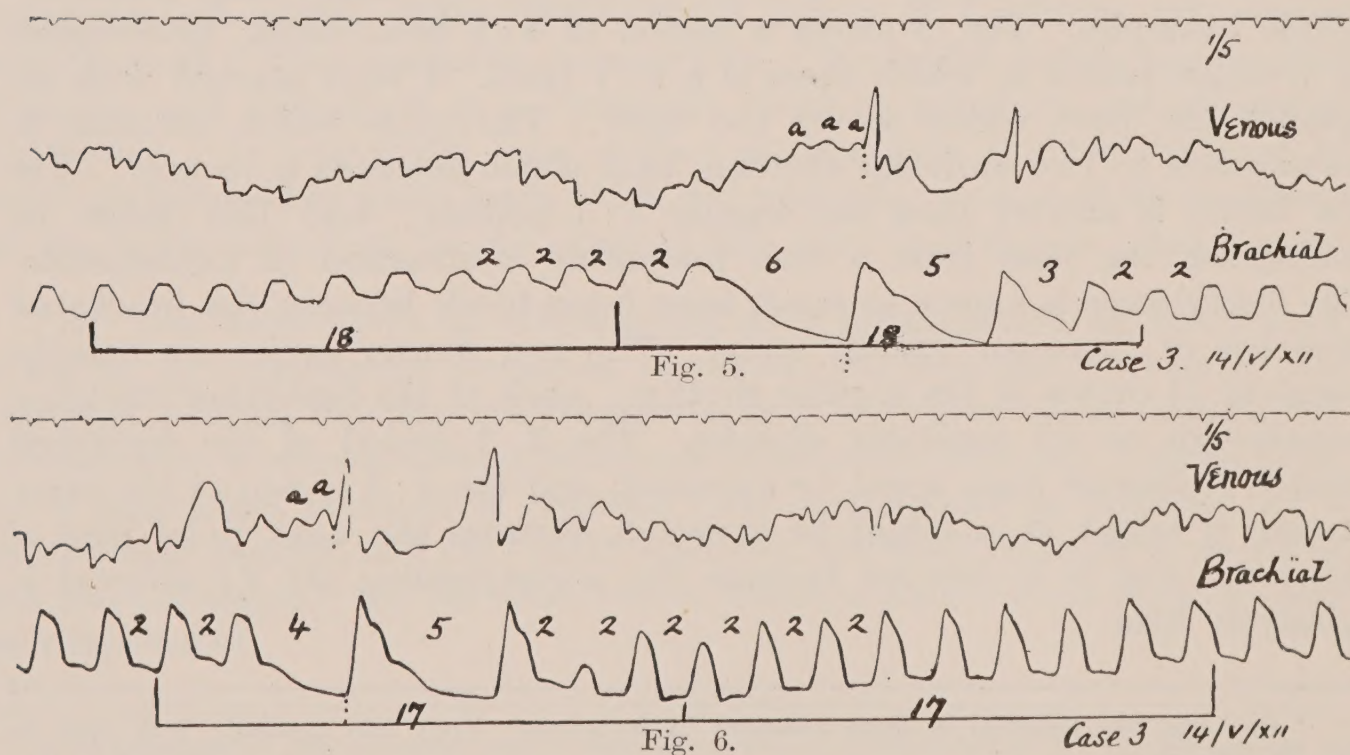


Fig. 5 and 6. Two polygraphic tracings showing arterial and venous curves, taken while the auricles were fluttering. They are published to show the methods of analysis as applied to arterial curves, and the general form of the venous curves,

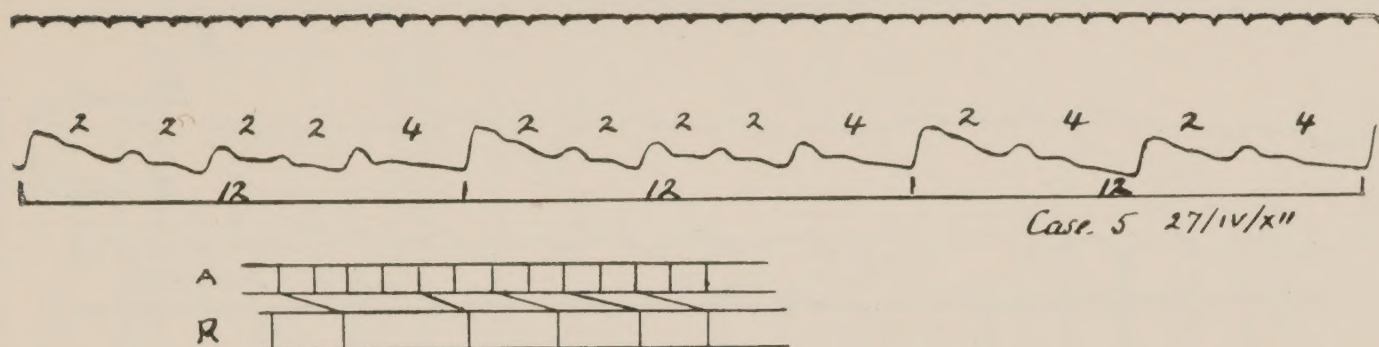


Fig. 7. An arterial curve from a case of flutter. The pulse is very irregular and bears a superficial resemblance to that of auricular fibrillation. Note the presence of alternation. An explanatory diagram, in which the relations of auricular contractions and radial upstrokes are illustrated approximately, accompanies this figure.

Fig. 4 shows the passage of a 4 : 1 period into a 2 : 1 period. Again the bracketed stretches are exactly equal; one corresponds to two 4 : 1 cycles, the other to four 2 : 1 cycles. It should be especially noticed that I have avoided the actual point of change in placing the brackets; and I have done so for the following reason. Bracketed stretches can be legitimately compared, only *when the points of each single bracket coincide with similar upstrokes*. That is to say, the beats at the points of a given bracket must be preceded by equal pauses; this is essential in all such measurements, otherwise the pulse beats in question will not have been equally delayed (bundle conduction and presphygmie interval will have been variable) and they cannot be taken as indices of the intervals which separate the corresponding auricular systoles. The first 2 : 1 cycle in Fig. 4 is longer than the rest; it is a strong beat and the auricular systole which was the cause of it took place at a shorter interval before it, than was the case with the succeeding beats. The last were all delayed in their appearance. Considering this first cycle of the faster rhythm, we have the choice of marking it as a 2 : 1 or a 3 : 1 cycle. Comparing it with the evident 2 : 1 cycles, it is shorter than $1\frac{1}{2}$ of these and longer than one of them. That a 2 : 1 cycle in this position would be relatively long is to be anticipated for the reasons stated, that a 3 : 1 cycle in this position should be relatively short is opposed to experience. Consequently, in the analysis of the remaining curves, when a cycle is longer than the calculated duration of x number of auricular cycles, such a cycle is marked x or $x + 1$ according to whether a long or a short cycle, respectively, precedes it. In adopting this system, I am merely following the well known rules of pulse analysis. That the rule is sound will be evident from a consideration of Fig. 2. Here I have marked the cycles according to this system where a stretch of a 2 : 1 ratio passes into an irregular stretch. If the cycles have been correctly marked, then the first seven cycles of 2 : 1 heart-block, which are bracketed, should be exactly equivalent to the next five cycles of the irregular period, which are also bracketed; for each bracketed stretch should correspond to fourteen auricular cycles. Such is actually found to be the case.

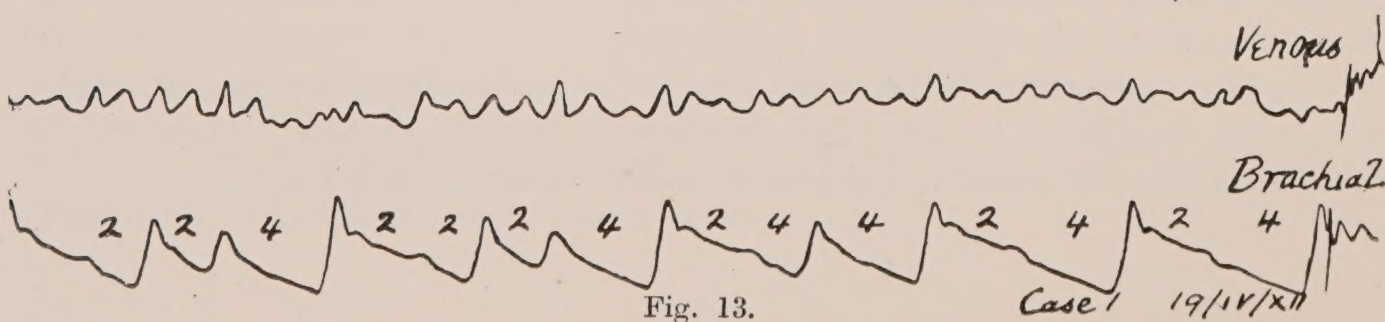
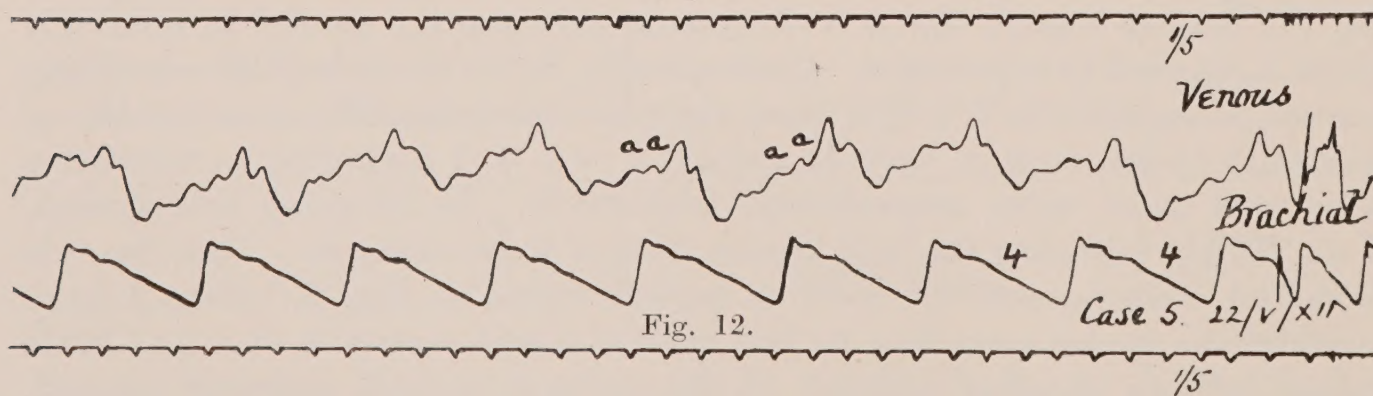
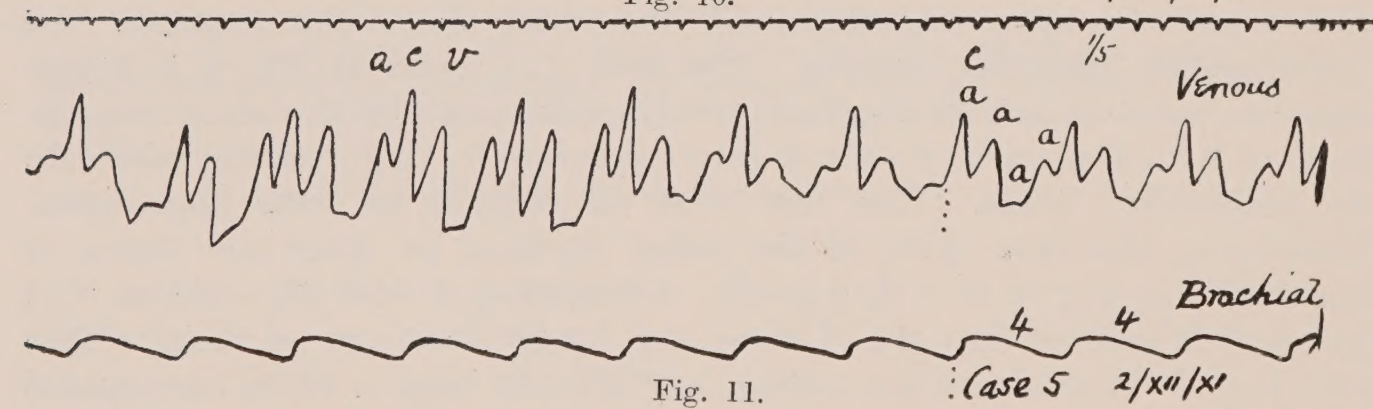
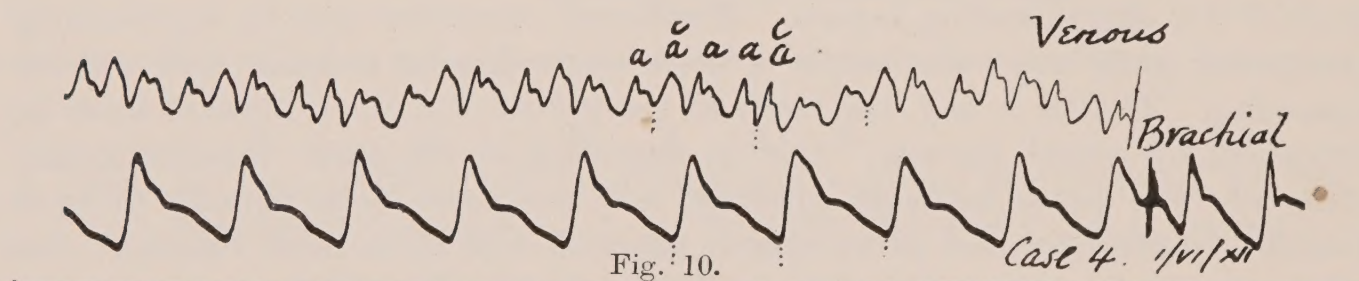
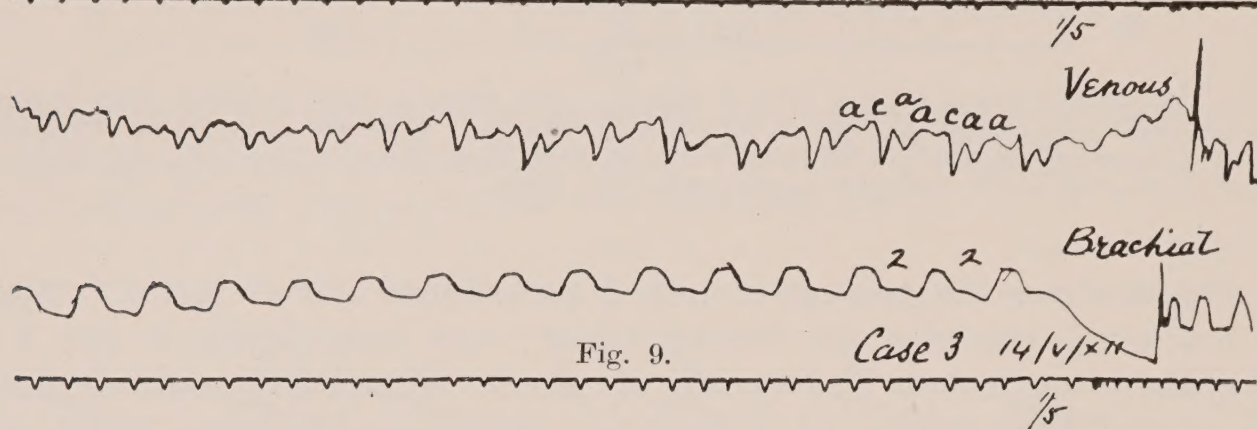
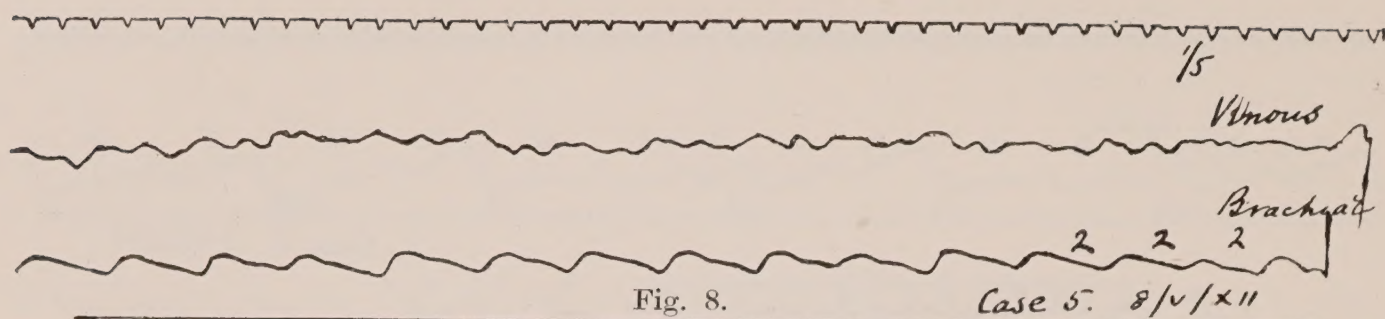


Fig. 8-13. Six polygraphic curves, showing the types of venous tracings which accompany auricular flutter.

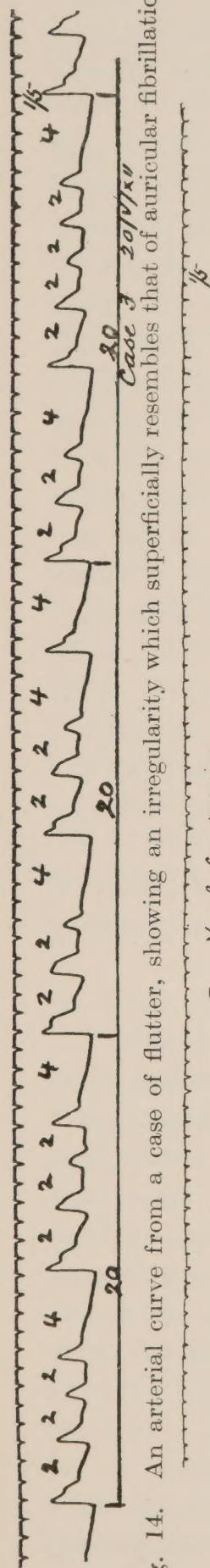


Fig. 14. An arterial curve from a case of flutter, showing an irregularity which superficially resembles that of auricular fibrillation.

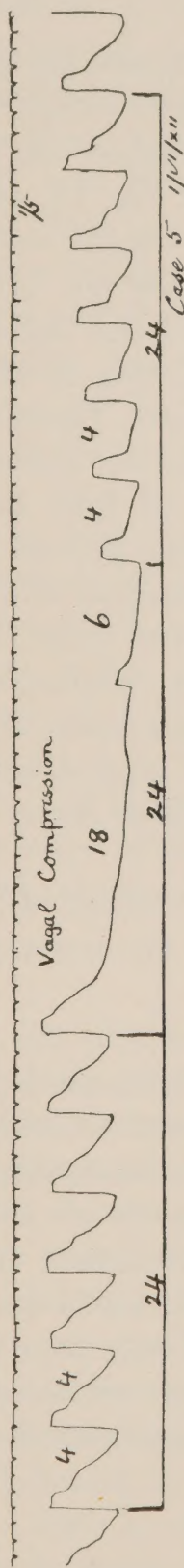


Fig. 15. An arterial curve showing the effect of right vagal compression during a period of 4:1 heart-block. The auricular rate is unaffected; the ventricular responses, after the long pauses, occur at expected intervals. Note the weakness of the returning pulse waves.

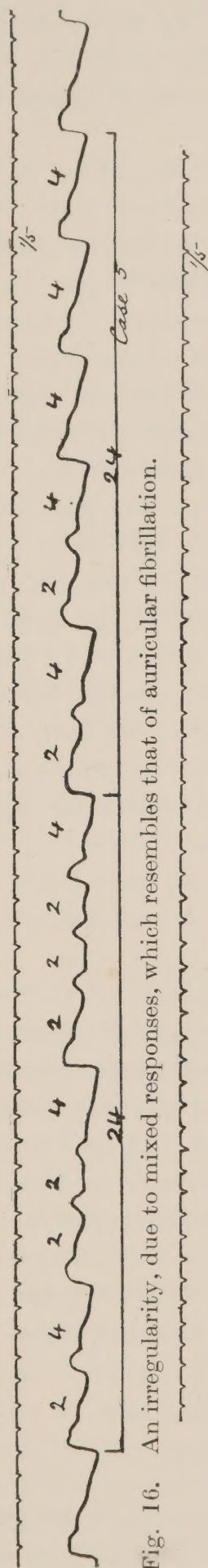


Fig. 16. An irregularity, due to mixed responses, which resembles that of auricular fibrillation.

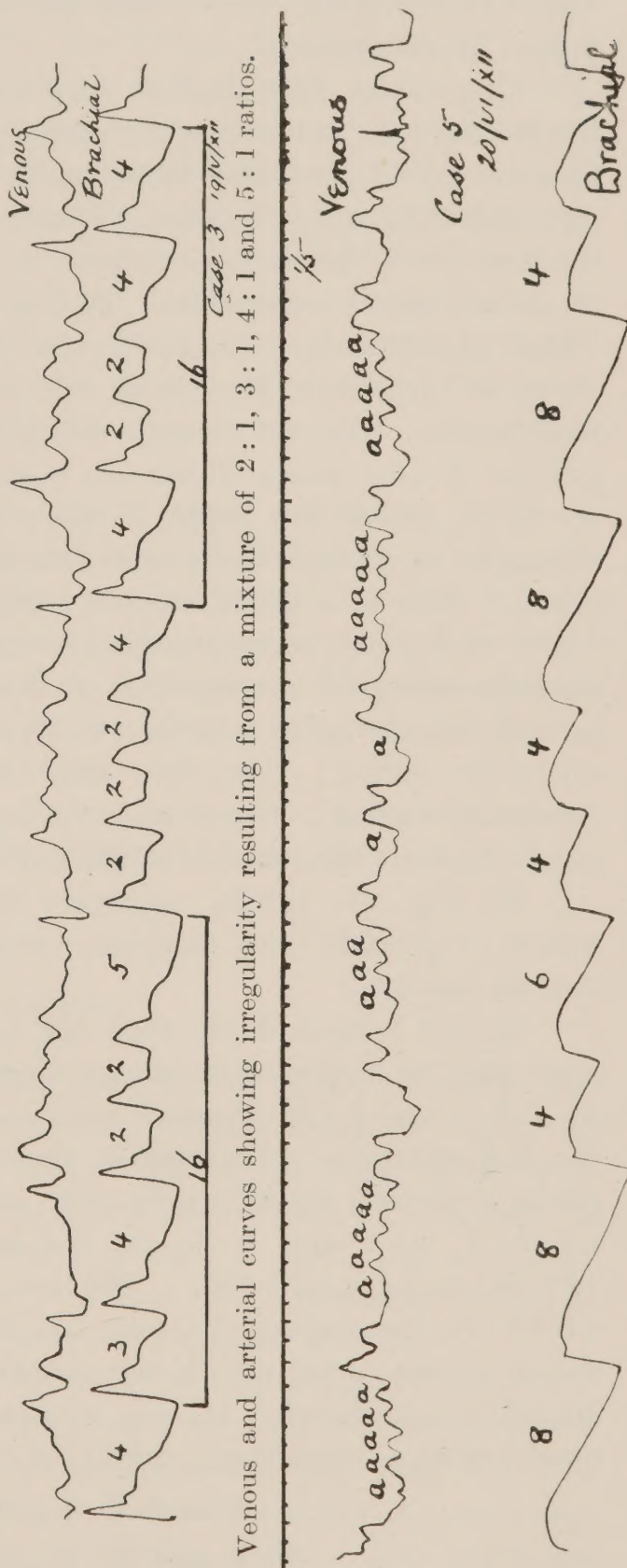


Fig. 17. Venous and arterial curves showing irregularity resulting from a mixture of 2:1, 3:1, 4:1 and 5:1 ratios.

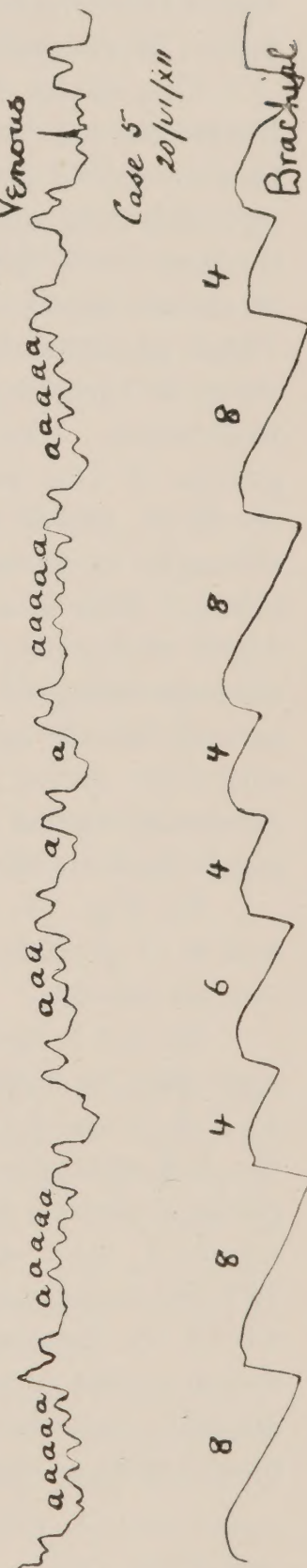


Fig. 18. Venous and arterial curves, showing irregularity as a result of a mixture of 4:1, 6:1 and 8:1 ratios.

Similar analyses are shown in Fig. 7, 13, 14 and 16, but in these figures the pictures are further complicated by the presence of conspicuous alternation; and in Fig. 13, certain of the beats are so weak that they barely affect the arterial curve; also the increased delay in certain beats of this figure is exceptional.

One of the chief reasons why the arterial curves accompanying auricular flutter should be thoroughly comprehended, is their possible confusion with the curves of auricular fibrillation. I have already indicated the manner in which they may be distinguished. The longer curves of Fig. 14, 16 and 17 illustrate this point especially. A cursory examination might easily lead to an erroneous conclusion. In Fig. 14, three equal stretches are bracketed. These are stretches of some length and the arrangement of the beats within them is variable. The pulse beats show groupings; batches of three and four occur. The curve may be split into shorter stretches. There are two groups of four beats which are equal in length, and there are three groups of three which are equal. Moreover the two groups of four beats are arranged in an exactly similar manner, as are also the three groups of three beats. This is a phenomenon which is never seen in auricular fibrillation, a fact which I have emphasised in my book.⁴ In Fig. 16, two stretches, each corresponding to twenty-four auricular cycles, have been bracketed. The stretch to the right may be subdivided into two, each equivalent to twelve auricular cycles; there are also three stretches exactly equivalent to six auricular cycles; three exactly equivalent to eight; and three exactly equivalent to ten, in this single curve.

In Fig. 18, similar analysis shows the presence of mixed 4:1, 6:1 and 8:1 periods; an analysis which is substantiated by the corresponding venous tracing.

So far I have dealt with the analysis of periods in which the ratio of auricular to ventricular contraction is as 2:1 or as simple multiples, *i.e.* 4:1, 6:1 and 8:1. In such curves, and in the absence of electrocardiograms, the full analysis is impossible, for the periods might be interpreted with perhaps almost equal justification as 1:1 periods, and the simple multiples, *i.e.* 2:1, 3:1 and 4:1, on the assumption that the auricular rates are 120-150 and not 240-300. We now come to the less frequent arterial curves, which by themselves disclose the true auricular rate. They are curves in which cycles occur which are exactly intermediate between certain of those already considered. If our choice lies between the interpretation of a mechanism as consisting chiefly of:—

2:1 and 4:1 periods or

1:1 and 2:1 periods,

then the appearance of a cycle of intermediate length may be of great value. Fig. 1 shows the interruption of 2:1 heart-block by an exceptionally long interval. It is almost exactly equal to two cycles of the faster rhythm; but it is not a 4:1 period; were that the case its length would be less, for

the large beat which succeeds it should have a short *As-Vs* interval. It is in reality a 5 : 1 period ; it is longer than the 4 : 1 periods of Fig. 14, which is from the same case. This analysis is completely justified by measurement of a stretch of curve taking in the whole disturbance. I have marked two equal stretches of curve ; the first contains six cycles of equal length ; the last contains one long cycle, and three and a half shorter cycles. If we adopt a simple analysis, and call the shorter cycles 1 : 1 periods, it is necessary to assume that, over the period of irregularity, the auricular rhythm has been disturbed, and disturbed in a peculiar manner ; somewhere an auricular cycle has been curtailed by exactly one half. The possibility of such a coincidence cannot be allowed. On the other hand, the whole curve is fully explained if we assume the higher ratio. In a series of auricular beats which might be labelled successively *a* and *b*, the responses before the disturbance are to *a*, the responses after the disturbance are to *b*. A parallel example is shown in Fig. 6.

From time to time, it happens that two of these *odd* ratios lie close together so that two may be taken into a single bracketed stretch. Fig. 17 is an example of this kind. Two stretches are bracketed in this figure ; each corresponds to sixteen auricular cycles. The second stretch is built up of *even* ratios, represented by 2 : 1 and 4 : 1 periods. The first stretch is built up of 2 : 1 and 4 : 1 ratios and, in addition, two *odd* ratios, represented by a 3 : 1 and 5 : 1 period, are interposed.

Thus it is evident that from time to time, where no 1 : 1 periods occur, full knowledge of the auricular rate may be obtained from a detailed analysis of arterial curves alone.

The venous curves.

The venous curves, when the auricles contract at extreme rates, are very varied in form, and the variability mainly results from the different degrees of heart-block which may be present. Where the heart-block is of high grade, be it complete or partial, as in Hertz and Goodhart's case or in Jolly and Ritchie's patient, and the ventricular rate is slow, the auricular waves may be distinct or even prominent.* When the ventricular action is fast, the auricular waves are usually indistinct or almost absent.

It is necessary that the forms of venous curves which accompany auricular flutter should be borne in mind, for not only are these curves often but imperfect guides to the identification of the condition, but they may actually mislead. This is especially so when patients first come under observation, for at such times the ventricular action is usually rapid. The auricular beats are close together, and the period of auricular diastole is of the shortest ; the ventricular systoles are also close set ; consequently, when a 2 : 1 ratio

* As a general rule too the auricular waves are more prominent as the rate of contraction is lower.

is present, each auricular contraction falls partly within the period of ventricular systole. In all probability there is considerable engorgement of the auricle as a result ; the weakness of its systole is thus readily accounted for. Whatever the explanation, the fact remains that when a regular 2 : 1 ratio is present, the venous curve usually consists simply of rapid and small undulations in which it is difficult, nay often impossible, to identify the expected waves with certainty. Examples of curves of this nature are seen in Fig. 5, 6, 8 and 9. When a regular 4 : 1 ratio is present, the *a* waves may be clear only towards the ends of the diastolic periods, as in Fig. 12. In rare instances of 4 : 1 or even a smaller ratio (*i.e.* 3 : 1 or 2 : 1) they may be distinct throughout (see Fig. 10 and the curves of Turnbull's and Rihl's cases). Sometimes the phlebograms are of such a form that they are readily misinterpreted ; in Fig. 11, I have marked what would appear as the obvious interpretation to the left (*a*, *c* and *v*) ; electrocardiograms taken on the same day demonstrated the correctness of the second interpretation, which is seen to the right of the figure ; the curve was taken while 4 : 1 heart-block was present. When long pauses occur between ventricular beats, signs of the auricular beats are sure to occur from time to time, as in Fig. 5 and 6 ; usually they are more evident than these figures suggest ; thus in Fig. 18, they are perfectly clear, and especially so towards the ends of diastoles. Each venous wave in Fig. 18 may be taken to represent an auricular contraction ; the least distinct is that which falls at the commencement of diastole.

General features of the electrocardiograms.

The electrocardiograms have been described already in illustrating the mechanisms presented by the separate patients of the series. It remains only to note certain features which are almost common to the curves of flutter and briefly to discuss these features.

The portions of the electrocardiograms which represent the auricular activity are peculiar in more respects than one. If we except the curves shown in Fig. 33, where the auricular rate is relatively slow, the auricular complexes in leads *II* and *III* are always contiguous ; that is to say, the separate electric complexes are so close together that the end of one marks the beginning of the next. The string is in constant motion ; there are no isoelectric stretches dividing one auricular electric cycle from the next. That the whole electric cycle corresponds to auricular systole is not possible, for if that were the case diastole would not be represented. It might be argued that diastole is so short that the isoelectric line, which we expect, is inappreciable and escapes observation. But this possibility may be excluded. The normal length of *P* is decidedly less than .20 sec. ; and if the auricular rate is fast *P* is shorter than this. If we measure the length of an auricular cycle in Fig. 31 we find it of approximately .22 sec. duration, despite the extreme rate of auricular contraction shown by the figure. The length of an

auricular electric cycle in flutter is inversely and exactly proportional to the rate. Thus, if the rates are 300 and 270, then the lengths of the individual cycles are $\frac{60}{300}$ and $\frac{60}{270}$. It follows that during auricular diastole electric potential is developed in the auricle. This observation is not an isolated one. The same fact has been noticed for the ventricle, when its movements are extremely rapid (see Levy and Lewis³). That potentials are developed in diastole has also been shown for a slowly beating ventricle. The *U* wave, originally described by Einthoven, lies in diastole.⁶ Although the electric curves may indicate the onset of auricular systole in flutter, they are but imperfect guides to the onset of diastole. It seems probable that, both in the case of the auricle and of the ventricle, chemical processes which occur in diastole are responsible for the string deflections. And as these diastolic deflections are always related to the commencement of diastoles, it seems natural to ascribe them to the more rapid reparatory metabolism which may be assumed to attend these phases of the cardiac cycles.

The auricular complexes in auricular flutter are remarkable for their constancy of form from case to case. Except again Fig. 33, and so far as curves have been published, the following statements are universally applicable. *P* in lead *I* is either a small and pointed summit, or is so insignificant that it cannot be found. *P* in lead *II* and *III* runs on either side into the adjacent complexes, so that a series of such complexes form a continuous wavy or zig-zag line; the auricular complex is found to be almost if not quite a purely convex summit wherever its onset can be defined.

The resemblance in shape and amplitude between auricular cycles from case to case is often so close that it suggests a distinct and special auricular mechanism. I draw particular attention to the forms of the complexes in Fig. 20, 29, 31, 32, 34 and 35, each figure being from a separate case. In all these the outlines are practically identical. There is a relatively abrupt upstroke and a relatively more inclined downstroke. The curves of *CASE* 8 are so similar to those of this series that it is unnecessary to publish them. The published curves of *CASES* 10, 11 and 14 were of the same form. Speaking of the curves of 13 cases in which electrocardiograms have been obtained, they conform to this type in no less than ten instances. If we compare the complexes in Fig. 28 and 29, it will be seen that the usual type is more distinctly assumed when the heart-block is of higher grade. Of the three remaining cases out of the thirteen, one (*CASE* 5) showed the usual type of complex during 4:1 heart-block stage; thus leaving two real exceptions (*CASES* 1 and 6 of the present series). In one of these (*CASE* 6) the auricular rate was relatively slow; in the other (*CASE* 2, Fig. 24) the divergence of type is not great.

It seems probable to me that this resemblance may largely result from the extreme auricular activity. The contiguity of separate auricular cycles is certainly to be assigned to this cause; if the further resemblances arise in similar fashion, then it should be capable of experimental proof. It is at least remarkable that in the single case where no resemblance can be traced

(Fig. 33) the auricular rate is decidedly less than in the remainder, namely 228. In *CASE 5*, the lowest auricular rate recorded electrocardiographically was 256, and of the curves for this day it may almost be said that contiguity is broken. It seems, therefore, that if contiguity is the result of great acceleration, such contiguity is fully established when the rate reaches 260 or surpasses it.

The acceleration of the auricles.

The lowest rate of auricular contraction in the series of collected cases was 200, the highest rate 330. A number of the cases have developed rates equal to or exceeding 300 (*CASES 1, 3, 5, 6, 10, 11, 14 and 15*). A notable quality of these accelerations is their relative constancy under different conditions. In *CASE 1*, the rate varied between 312 and 324; in *CASE 2*, between 266 and 289; in *CASE 3*, between 264 and 324; in *CASE 4*, between 260 and 278; in *CASE 5*, from 244 to 300. And these counts are of rates found over long periods of time and under very varying circumstances. From day to day the correspondence of rate is often remarkable (see especially *CASES 4, 3 and 5*). The changes of rate with posture are almost negligible; in *CASES 2 and 3* pulse counts were taken and no differences were noted. In *CASES 4 and 5* counts were made from pulse curves; the ventricular rate was less than three beats faster per minute in the standing position in one case, and no constant differences could be found in the other.*

In two of my patients, electrocardiographic records were often taken shortly after they came to the school; they walked some distance; other records were taken after they had rested. In none of the records taken on any one day can an appreciable change in auricular rate be found. In two patients (*CASES 4 and 5*) continuous records of the pulse (during periods of 2:1 ratio) were taken, commencing directly after exercise; little or no change of rate could be detected. In three patients in which counts could be made, pressure upon the vagus, sufficiently strong to stop the ventricle for periods of several seconds, had absolutely no effect on the auricular rate. Rihl noticed the same fact. The fast rhythm appears to be almost uninfluenced by conditions which materially change the rate of the normal pulse. I have repeatedly noted the same stability of rate in cases of paroxysmal tachycardia of lower grade, and have spoken of it in previous papers. Rihl has also been impressed by it in flutter, and has concluded from this observation that the acceleration is due to over-action of the sympathetic, while the vagus influence is entirely in abeyance. But this view is one which, it seems to me, has insufficient facts to support it; and my belief is dictated by the following arguments.

* In all cases the observations were made during periods of 2:1 ratios.

It is necessary to bear in mind that we are dealing with a heart action which is very probably ectopic in nature ; it may not be derived from the normal pacemaker.* The reason for regarding the rhythms as ectopic is that the auricular complexes of the flutter period differ so essentially from those of the normal period ; the comparison can be made in the curves of my first article (*CASE 5*). It may also be made in the curves of *CASES 1, 2, 14, 15 and 16*. When my first article was written, I was more definitely of opinion that the new rhythms of flutter are ectopic, and for the reason which influenced Rihl. The auricular complexes were read as inverted complexes. Further examination of a more extended series of curves shows that this is almost certainly not the case. The divergence from the normal is therefore actually less than it was considered to be ; nevertheless it is considerable. Whether it can be ascribed wholly to change of rate remains to be proved ; but I think that change of rate is not the sole reason, after comparing the complexes of the flutter and of premature beats in the same case (*CASE 5*) ; the latter are undoubtedly ectopic and are not dissimilar to the complexes of the flutter.

So long as we are in doubt, the possibility that the rhythms are ectopic remains and should be taken into account, and this more especially because, if ectopic, the special nerve relations can be explained. Tachycardias of lesser rate show the same failure of reaction to nerve influences, as I have pointed out. If one class (flutter) is to be explained by overaction of the sympathetics, so also must the other, if Rihl's arguments are permitted. These slower tachycardias can often be demonstrated beyond doubt to be ectopic. In the slower tachycardias of auricular origin the absence of nervous control may be ascribed to the altered anatomical relation between nerves and pacemaker. We are not justified in assuming anything in regard to the innervation of new centres of impulse formation until more experimental facts have been obtained. I am aware that in certain experiments, notably those of Rothberger and Winterberg, in which the auricles were under the influence of poisons (BaCl_2 , &c.) sympathetic stimulation led, not only to a dislocation of rhythm, but also to acceleration of it. It remains to be shown that the conditions are comparable to those now studied. At all events, because the heart rate is greatly accelerated it does not follow that the sympathetic nerves are in any way to blame. Extreme accelerations of the ventricle may be induced by ligation of a coronary artery or a small branch of such a vessel ; and such tachycardias are evidently the result of nutritional disturbances of intracardiac origin ; they have nothing to do with the extrinsic cardiac nerves. It may be that in many instances of tachycardia the nerves of the heart ultimately provoke the new rhythm ; such a statement cannot be denied ; neither in the present state of our knowledge can it be justly affirmed. We know that in the normal heart, new and rapid rhythms cannot be provoked by such stimulation. It is

* Rihl recognised this possibility, but his reasons for a conclusion to this effect are not convincing, neither, if it be true, does he appreciate its full significance.

necessary that the organ should be brought first into a curiously sensitive condition, whether by the injection of adrenalin, the chlorides of barium or calcium or by chloroform or perhaps by simultaneous vagal stimulation, before sympathetic excitation is successful. The question is an important one, for so long as we accept the view, adopted as I think prematurely by Rihl, so long shall we look for the cause of the whole disturbances in the central nervous system; whereas it seems to me, our first endeavour should be the exclusion, at all events, of a primary cardiac defect. There are clear connecting links between these extreme accelerations and premature contractions and slower tachycardias on the one hand, and fibrillation of the auricles on the other. These disturbances are associated in most instances with distinct evidences of cardiac lesions; and there is every reason to believe that the extreme accelerations with which we are now dealing are of similar origin. All appear to be part and parcel of the same process, namely, heterogenetic* impulse formation in the heart. It is clear that such impulse formation can result apart from all nerve influences (*i.e.*, in obstruction of an artery); it is by no means so clear that it can result from morbid nerve influences playing upon a perfectly healthy organ.

The response of the ventricle.

Patients in whom there is an auricular action exceeding 200 per minute usually present a slower ventricular rate, as illustrated by the present series of cases. Of the sixteen cases in which graphic records of auricular flutter have been obtained, all have shown heart-block from time to time and it has been either constant or present on most occasions; the lowest grade of block has usually been a 2:1 ratio, or response of the ventricle to each second auricular impulse. It is in this condition that the patients usually come under observation for the first time. That lower rates may occur while no drugs are being administered will be evident from Jolly and Ritchie's report (*CASE 10*), in which complete-heart block was present both before and after the onset of the flutter; and also, though less clearly, from Hertz and Goodhart's case,¹⁴ for here the responses were at wide intervals; but this case was complicated by the presence of premature ventricular contractions. When the ventricle is beating at half the rate of the auricle, and the latter contracts 300 times in the minute, it may be asked whether the tissues joining auricle and ventricle are functionally defective? This question appears to be answered by the occasional occurrence of a 1:1 response in the same cases; rates of 290 and 300 were reached by the ventricle and were recorded by Mackenzie (*CASE 14*), and it is more than probable that 1:1 response occurred from time to time in *CASE 6*. It may be concluded that the junctional tissues can conduct impulses which succeed each other at this rate and that the ventricle can respond to them. When 2:1 heart-block is seen, it may be said therefore that there is a primary

* This term has been fully described in my book "The Mechanism of the Heart Beat."

deficiency of conduction, but this deficiency is probably slight, for acceleration of the auricle tends to exaggerate the degree of heart-block, as has been clearly shown experimentally.

While discussing the electrocardiograms, it was pointed out that the auricular representatives are as a rule convex. The *P* summits, which are contiguous, commence in upstrokes, a fact which was deduced from their form in lead *I*. If we measure the length of the *P-R* interval in this lead in Fig. 28, it is found that it amounts at the most to .1 sec.. Considering that 2 : 1 heart-block is present, this interval is much shorter than might have been anticipated ; and its brevity suggests that the ventricle responds to the preceding auricular contraction. That such is the case is demonstrated, I think, from an examination of electrocardiograms which show irregular ventricular action in the same case. Fig. 36 exhibits a single 5 : 1 period which is followed by successive 2 : 1 periods. Consider the upstrokes of *P* which precede the second and third *R* summits in this photograph. The first lies further from the corresponding *R* summit than does the second. If the two ventricular beats in question were each responses to the preceding auricular summits, then the *As-Vs* interval of the first cycle would be the greater ; but this *As-Vs* interval follows the longer instead of the shorter pause. We know from the arterial curves that the *As-Vs* interval is varying in the ordinary manner during periods of irregularity ; that is to say that the shorter pauses are succeeded by the longer *As-Vs* intervals. It must be also the case in this figure. If the first ventricular beat is a response to the preceding auricular contraction, then the second is a response, not to its preceding auricular contraction, but to that which is further removed from it. Such is the interpretation which has been marked upon the figure. The remaining cycles of the figure are all of 2 : 1 ratio, and as the *Vs-Vs* interval shortens gradually for the first few beats, so the *As-Vs* interval is seen to increase slightly. As a result the auricular contraction which gives rise to a ventricular response is eventually placed so that its upstroke coincides with the commencement of the preceding *R* summit. In this figure the reason why the first cycle of a 2 : 1 period is relatively long is clearly seen ; its unusual length depends upon the arrangement of *As-Vs* intervals. The figure is also of interest because it shows alternation, which commences with the third of the rapid arterial upstrokes. It does not commence with the second, as is usual, for the reason that a longer pause precedes this beat.

Another example of simultaneous arterial and electrocardiographic curves is shown in Fig. 37. This was taken from another case (*CASE 5*), and the ratios for the separate cycles are clear. A bigeminal action, consisting of 2 : 1 and 4 : 1 periods passes into a simple 2 : 1 heart-block. The figure illustrates to perfection the impossibility of accepting the interval between adjacent *P* and *R* summits. It is known from the electrocardiograms given by lead *I* in this case (for examples, see previous article), that the upstrokes represent the commencements of auricular systoles.

If we take those upstrokes in Fig. 37 which directly precede *R* summits, the *As-Vs* interval decreases after the longest pauses to less than .07 secs.,* and the greater part of the auricular systole is buried in the ventricular systole. The responses must therefore have been to auricular systoles which were further removed, as illustrated by the diagram drawn on the figure. Thus it seems clear that in pure 2 : 1 curves, the responses are as marked upon Fig. 28 ; it is also evident that two auricular systoles may occur, and that before the first of them is responded to by the ventricle, the second auricular systole may be almost completed ; it is also obvious that two auricular impulses may be travelling along the junctional tissues at the same moment.

The deficiency, when a 2 : 1 ratio is present, is attributable to pathological change rather than to auricular acceleration pure and simple ; the hearts in which flutter occurs are abnormal. The demonstration of imperfect conductivity, which occurs so constantly in these cases, is of considerable importance. Clinically and pathologically the cases are closely allied to those which exhibit auricular fibrillation, and they warn us most clearly against the conclusion that in auricular fibrillation all the impulses are transmitted. Later we shall see that there is good reason for believing that the contrary conclusion is more justifiable.

Cases of auricular flutter are of importance because they permit exact observations upon changes in conduction in the human heart. One factor is practically constant, and so far as we know it is constant in no other condition,† the auricular impulse rate is unvarying. Constancy of the auricular impulse rate is important because, as I have stated, variations in auricular rate in themselves alter conduction. Observations upon heart-block, where the normal rhythm is present, are not pure, because this factor has always to be taken into consideration. In all the cases which have come under my observation, nothing has been more fully established than that exercise and excitement immediately increase the rate of the ventricle, the rate of the auricle remaining practically unaltered. I have made a great many observations from this point of view. In *CASES* 3, 4 and 5, rising from the lying to the sitting or standing posture would almost invariably and immediately change a 4 : 1, or irregular response, to the original 2 : 1 response. In *CASE* 3, this effect was most conspicuous. While this patient was upon digitalis or strophanthus he often walked as far as the school. When he arrived, the ventricular contractions were always regular and at a half the auricular rate. If he lay for a few minutes the pulse became irregular and slower, and mixed responses (2 : 1, 3 : 1, 4 : 1 and 5 : 1) were then observed. Often he sat with his arms and foot lying on the electrodes preparatory to the taking of curves, and the regular and fast ventricular rate would be

* A time-marker is not shown in this figure but the auricular rate was calculated on the same day in other curves to be 272 per minute. The *P* summits are separated therefore by intervals of .22 secs..

† It is possibly constant in auricular fibrillation.

maintained ; on many occasions if he raised the right foot and rested it upon a chair a slower and irregular action immediately appeared. And it would disappear and reappear with great constancy according as the leg was in the more strained position, while sitting, or horizontal. The reaction was an extremely sensitive one. If I wished to obtain arterial curves of the irregular periods, it was usually insufficient to strap the receiver to the outstretched arm, the arm required support before the pulse became slow. The tension in the upraised arm was almost always sufficient to establish temporarily the fast and regular action of alternate responses. A few minutes conversation, or an unexpected visit, always provoked a higher ventricular rate. The rate was almost always faster after meals ; swallowing never seemed to affect it in this or the other patients. When the patient lay quiescent and the heart beat irregularly, breaking from regular 2 : 1 periods into irregular periods of higher ratios, the reason for the individual changes could not be ascertained. Breathing had no relation to them.

In all those cases of this series in which digitalis or strophanthus was administered, the original grade of heart-block was increased. Hearts in which the auricles are in a state of flutter appear to be peculiarly susceptible, as are those in which the auricles are fibrillating, to the action of drugs of this group. This susceptibility is largely attributed to the rapidity with which impulses are showered upon the junctional tissues. A condition of 2 : 1 block is rapidly converted to one in which there are mixed periods of 2 : 1 and 4 : 1, or 2 : 1, 3 : 1, 4 : 1 and 5 : 1. Eventually in most cases 4 : 1 heart-block is established. The block may increase further in some instances (see *CASE 5*) and 6 : 1 and 8 : 1 periods may be observed. Personally I have not employed atropine, but Rihl has used it on several occasions in one patient ; where digitalis heart-block is present atropine restores the original ventricular rate, according to this writer. It appears therefore that the action of digitalis in this instance was chiefly if not entirely an indirect one through the vagi.

Rihl found that pressure on the carotid sheaths produced slowing of the ventricle (an increase of block) in his cases. The same observations have been made repeatedly in three cases of my own series (*CASES 3, 4 and 5*). In each instance pressure upon the carotid sheath increased the grade of block ; the vagi appeared to be particularly sensitive to even light pressure and in one case (*CASE 5*) the application of the jugular receiver to the neck on certain days was almost always followed by slowing of the ventricle. It was a matter of indifference as to which side was pressed upon ; the effect was equally conspicuous upon the left as upon the right side. In the three cases in which I have tested the effect of vagal pressure, the procedure gave a positive result more readily when the patient was under the influence of digitalis or strophanthus. Control observations were made in two cases (*CASES 4 and 5*) ; in one (*CASE 5*) patient I could obtain no effect at all even with the deepest pressure while the patient was uninfluenced by drugs ; in the other 2 : 1 heart-block was temporarily converted to irregular responses

at 3 : 1, 4 : 1 and 5 : 1 intervals. It is not evident from Rihl's paper whether his patients were under drug influence when the observations were made, except in one instance when the patient was upon digitalis. There was no demonstrable effect upon the auricular rate on any occasion ; the result was always a pure conduction effect. These results are of importance because they suggest the control of the junctional tissues by both vagal nerves in the human subject ; and because they fail to substantiate a view that there is a preponderating influence of one nerve over the other. Examples of the curves obtained are shown in Fig. 15 and in an electrocardiogram of my previous article.

A study of the reaction of the ventricle to the rapidly beating auricle reveals an interesting relation between the rates in the two chambers. There is a remarkable tendency for the ventricle to respond so that the ratio is an even one. 2 : 1 heart-block is the commonest relation of any, the only other ratio which at all commonly gives rise to a regular ventricular action is a 4 : 1 ratio. On one occasion and on one only, the ventricle has beaten in response to each third auricular stimulus, and then only for short spaces of time. When the ventricle is irregular, the irregularity usually consists of mixed 2 : 1 and 4 : 1 periods. Sometimes, it is true, isolated 3 : 1 and 5 : 1 periods appear ; but they are rare by comparison. In the single instance in which a higher grade than 4 : 1 heart-block was seen (*CASE 5*) the longer pauses consisted of 6 : 1 and 8 : 1 periods. Even ratios are far too common to be accounted for by coincidence, though it must be acknowledged that an explanation of the phenomenon is difficult. The fact remains that when the ventricle is slowing in these cases, so as ultimately to beat at a fourth of the auricular rate rather than a half, it tends to pass into a condition in which 2 : 1 and 4 : 1 periods are mixed and not to exhibit regular 3 : 1 ratios. Samojloff has drawn attention to the similar method of response of the frog's heart, in which conduction disturbances have been induced experimentally.

The relation of auricular flutter to other morbid auricular mechanisms.

In an article written in conjunction with Dr. Schleiter, I have already drawn attention to the close relation between paroxysmal tachycardia of auricular origin and auricular fibrillation. The conclusion that they are part and parcel of the same pathological process was supported by a number of facts, none of which were more noteworthy than the frequent occurrence of the two conditions in the same patient, and especially the immediate passage of one to the other. The argument applies even more strongly to auricular flutter, for in thirteen cases of the present series auricular fibrillation occurred in seven, and in six, one condition passed directly into the other.* The separate mechanisms must be very closely related ; and it is probable

* The actual transition was seen in one case only (*CASE 14*), and in this one a number of such transitions were recorded.

that they have a similar pathogeny. The occasional occurrence of tachycardia of a more simple form (*CASE 2*), and the frequent occurrence of single premature beats in the same patients when a sinus rhythm has been re-established (they were present in five out of the six cases in which the normal mechanism was observed) seems to connect flutter with the remaining auricular mechanism in which disturbing beats are found.

The conclusions that

1. Single premature auricular contractions,
2. Small groups of the same,
3. Paroxysms of tachycardia from single auricular foci,
4. Auricular flutter,
5. Paroxysms of tachycardia from two or more auricular foci, and
6. Auricular fibrillation

arise essentially in the same manner, namely, through the pathological or heterogenetic origin of new impulses in the auricle, are clearly suggested by the facts at our disposal.

The number of impulses which reach the junctional tissues in auricular fibrillation.

The passage of regular tachycardia of auricular origin into auricular fibrillation permits a theoretical comparison of the number of impulses which reach the junctional tissues in the two conditions. It is known from experiment that an acceleration of the auricles in the presence of a slight grade of heart-block tends to reduce the ventricular rate, and that the greater the auricular acceleration, the slower is the action of the ventricle. In the past we have had no data which have permitted us to gauge the rate at which impulses reach the junctional tissues when the auricles fibrillate. The view that all pass in experiment is but pure assumption. It may be that each second or each third impulse is transmitted, or that they are carried irregularly. That all pass in clinical cases is certainly not the case, for while in experiment the rates of the ventricular responses are always high, in patients they are variable and may be very low. We may argue that if the heart-block is present and the auricular flutter passes into fibrillation and the rate of the ventricle is reduced, the impulses are more frequent in the last named condition. We may make this comparison of rates in several cases of the series. In *CASE 1* the rates of auricle and ventricle before fibrillation appeared were 324 and 81, respectively. At the onset of fibrillation the ventricular rate was 79. In *CASE 2*, the auricular rate varied between 266 and 333; at the onset of fibrillation the pulse rate fell from 70-96 to 48-68. In *CASE 5* the auricular rate was 288-300, and at the

onset of fibrillation the pulse rate fell from 72 to 44. In Mackenzie's case (*CASE* 14) the auricular rate was 280-300 and at the onset of fibrillation the pulse rate fell from 140 to 55. Thus the ventricular rate becomes slower when fibrillation commences, or exceptionally, remains the same. It may be said of course that the heart-block resulting from digitalis has increased; but this seems improbable, for in most cases the degree of heart-block has been practically stationary for several days upon fixed doses of the drug. So far as these observations can be taken as evidence, therefore, it appears that the rate at which impulses reach the junctional tissues is greater while the auricles fibrillate than when they are in a state of flutter. The transitions suggest that a large proportion of the fibrillation impulses are always blocked in man even when ventricular rates of 200 are reached, and that the actual number of such impulses surpasses 270 or 300 per minute.

Digitalis as a therapeutic measure in auricular flutter.

In a previous article I recorded the passage of flutter to fibrillation during digitalis administration. A similar reaction was recorded in cases which, as it now appears, were of the same nature by Mackenzie and Turnbull. In the present paper I have added two further instances. In my original case the normal rhythm was resumed shortly after digitalis was withdrawn. The same phenomenon was witnessed in two cases which are now recorded (*CASES* 1 and 2). The normal rhythm was also resumed in Mackenzie's case and Turnbull's patient. It appears consequently that the production of fibrillation by digitalis administration is an important therapeutic measure in cases of flutter. The flutter may have lasted as long as four or five months (*CASES* 2 and 15); in fact, it is usually a persistent condition; it may last for three years or more; fibrillation has abolished it for observed periods, varying from a few weeks to several years. The induced fibrillation is a temporary event, and at its offset it is not the original tachycardia but the normal rhythm which is restored. From these facts it seems likely that whatever the final cause of the flutter, it is not a persistent one and that its continuation for long periods results from a perverse habit which the auricles acquire. Fibrillation seems to act by submerging the original fast rhythm. During the course of experiments on animals, regular tachycardias which are induced by stimulating some portion of the heart wall sometimes persist; they may be abolished by inducing a tachycardia of a faster rate by stimulating another point; and when this second stimulation ceases, the normal rhythm is restored. The induction of fibrillation is equally effective in these circumstances. Fibrillation presumably acts in a similar fashion in clinical auricular flutter, and this is a view which may be held to support the conclusion that the impulses arising in fibrillation and reaching any single point of the auricular tissues (such as the point at which flutter originates or the outlet to the ventricle) are more numerous than those of flutter itself. The hearts which manifest

flutter of the auricle are especially susceptible to digitalis and strophanthus in another way. As has been recounted, the grade of block is almost always increased in the earlier stages. If the full reaction and the return to the normal rhythm cannot be achieved, at all events the ventricular rate may be reduced and the heart obtains rest.

Summary and conclusions.

1. A condition is described and is spoken of as “auricular flutter.” It is not uncommon clinically (sixteen cases are collected), and it occurs for the most part in elderly subjects. It is characterised by an extremely rapid auricular action; the rate being from 200 to 330 per minute (usually at about 300 per minute). Generally, when it develops, it persists for months or years; it may occur in shorter paroxysms.

2. The auricular rhythm is the result of pathological or heterogenetic impulse formation. The new rhythm is probably also ectopic. It is not under nerve control. The rate is wonderfully constant in most subjects and is practically uninfluenced by posture, exercise or nerve stimulation. Its persistence is probably attributable to habit rather than to a continuation of the exciting cause.

3. As a rule the ventricular rate is one half the auricular, but it may be the same as the auricular, or any grade of heart-block may be present. Thus the ventricle may beat at rates varying from 30 to 300; it may beat regularly or irregularly. The rate of the ventricle is controlled by the functional condition of the junctional tissues; conduction is decreased by digitalis and its allies, and increased by exercise. The heart-block is increased by vagal compression, and this seems to be especially the case while it is influenced by digitalis.

4. When 2:1 heart-block is present the *As-Vs* interval may be so long that the auricular summit *P* of one cycle may fall with the *R* summit of the preceding cycle. Two auricular impulses may be travelling towards the ventricle at the same time; the ventricle may not respond to an auricular impulse until the next auricular systole is almost completed. The ratio of auricular and ventricular rates is generally an even one. Odd ratios when they occur are usually solitary.

5. Auricular flutter is closely related to similar tachycardias of lesser rate on the one hand and to auricular fibrillation on the other. It may pass to one or the other. All such disturbances have a common pathology.

6. Impulses probably reach the junctional tissues in auricular fibrillation at rates exceeding 270 or 300 per minute.

7. Even when it has been present for many months, flutter may often be abolished by the administration of digitalis. This drug induces temporary fibrillation, and the normal rhythm is subsequently restored and may persist. But the administration of digitalis may be advisable, even when this full reaction cannot be obtained, for the purpose of reducing the ventricular rate.

8. The arterial curves when the auricles are in a state of flutter, are of very varied form. The pulse may be perfectly regular and slow or fast. Pulse irregularities which suggest the presence of extrasystoles or fibrillation are frequent. Often the full diagnosis may be made from these curves alone.

9. The venous curves are almost always obscure, on account of the weakness of the auricular systoles. When the pulse pauses are long the *a* waves may be distinct. Certain of the venous curves give an erroneous impression that a normal rhythm is present.

10. The electrocardiograms show auricular complexes which are contiguous at rates of 260-335 per minute. The line of curve never runs isoelectrically during ventricular diastole. As a rule, two auricular summits are seen for each ventricular, but under these circumstances alternate auricular representatives are easily overlooked. The auricular summits in lead *I* are small and pointed. In leads *II* and *III* they are extremely prominent, being equivalent as a rule to 2×10^{-4} or 3×10^{-4} volts; they may reach an equivalent of 4×10^{-4} volts; in these leads the complexes are convex. Together they form a continuous wavy or zig-zag line. The form of the *P* summits is very fairly constant from case to case, and the resemblance is often so exact as to suggest a definite and specific mechanism.

BIBLIOGRAPHY.

- ¹ HERTZ AND GOODHART. Quart. Journ. of Med., 1908-9, II, 213.
- ² JOLLY AND RITCHIE. Heart, 1910-11, II, 177.
- ³ LEVY AND LEWIS. Heart, 1911-12, III, 99.
- ⁴ LEWIS. "The Mechanism of the Heart Beat," London, 1911.
- ⁵ LEWIS AND SCHLEITER. Heart, 1911-12, III, 173.
- ⁶ LEWIS AND GILDER. Phil. Trans. roy. Soc., 1912, CCII, 351.
- ⁷ LEWIS. Heart, 1911-12, III, 279.
- ⁸ MACKENZIE. Heart, 1910-11, II, 378.
- ⁹ RIHL. Zeitschr. f. exper. Pathol. u. Therap., 1911, IX, 496.
- ¹⁰ SAMOJLOFF. Archiv. f. Anat. u. Physiol., 1907, Phys. Abth., Sup. Bd., XXIX.
- ¹¹ TURNBULL. Heart, 1911-12, III, 89.

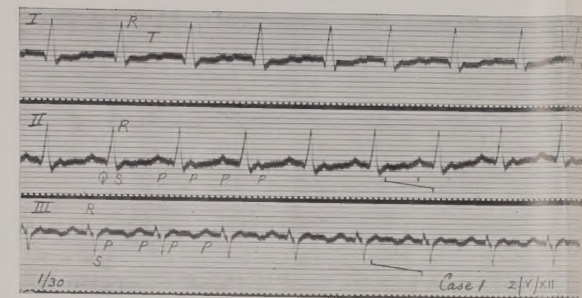


Fig. 19.

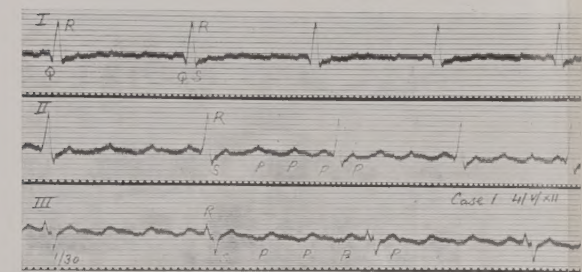


Fig. 20.

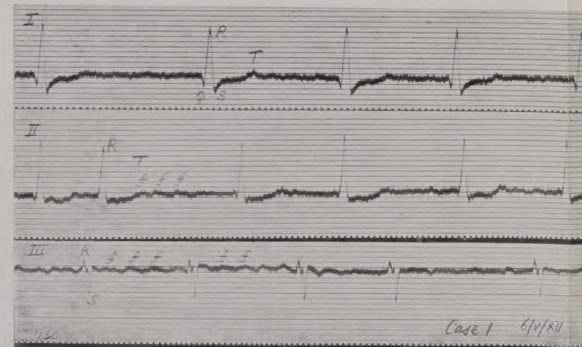


Fig. 21.

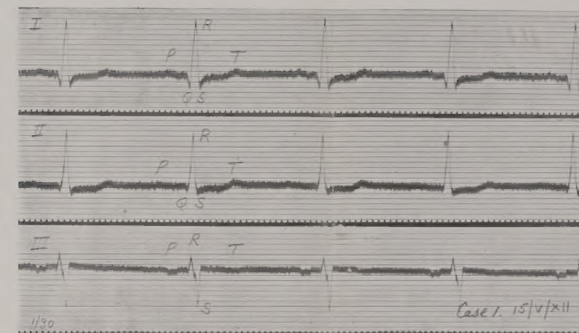


Fig. 22.

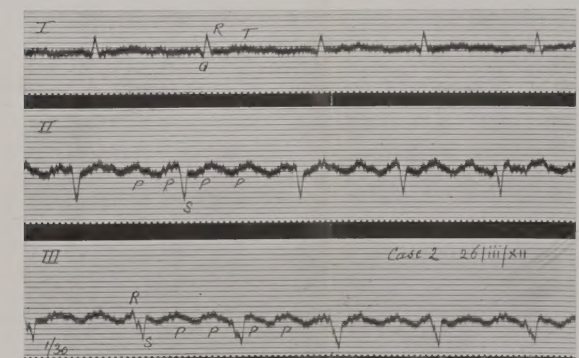


Fig. 23.

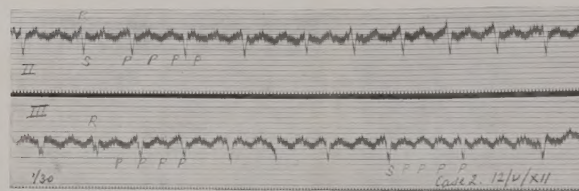


Fig. 24.

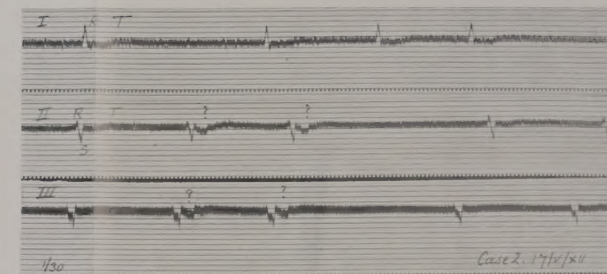


Fig. 25.

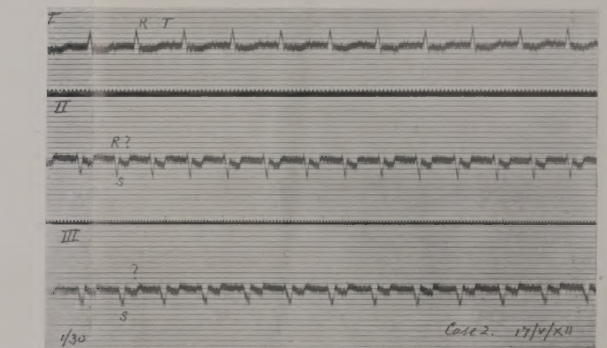


Fig. 26.

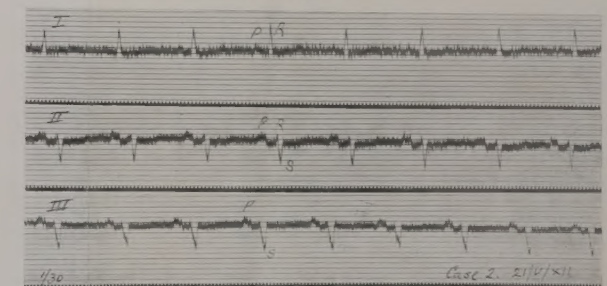


Fig. 27.

Fig. 19-22. A series of four curves showing the changes in mechanism which occurred in a case of auricular flutter under digitalis therapy. The dates upon which curves were taken are marked upon these and subsequent figures. The three leads *I*, right arm to left arm, *II*, right arm to left leg, and *III*, left arm to left leg are marked, and are arranged in this order from above downwards in each figure. The standard in all figures where horizontal lines are present is the usual one; 1 cm. corresponds to 1/1,000 volt. The time is in one-thirtieth sec..

Fig. 19 shows 2:1 heart-block.

Fig. 20 shows 4:1 heart-block.

Fig. 21 shows auricular fibrillation.

Fig. 22 shows the normal rhythm.

Fig. 23. Electrocardiograms from another case, showing 3:1 responses.

Fig. 24-27. A series of curves from the same case showing the response of the heart to digitalis.

Fig. 24 shows 2:1, 3:1 and 4:1 responses.

Fig. 25 shows auricular fibrillation.

Fig. 26 shows a curious form of tachycardia in which the auricular representatives are not seen.

Fig. 27 shows the normal rhythm.

Fig. 28 and 29. Electrocardiographic curves from a case of auricular flutter. The second figure shows the effect of digitalis administration.

Fig. 30 and 31. Electrocardiographic curves from a case of auricular flutter. The second figure shows the result of digitalis administration.

Fig. 32 and 33. Electrocardiographic curves from two cases of auricular flutter.

Fig. 34. Republished from Turnbull's paper, after modification. *I*, *II* and *III* are from the three leads during the period of flutter; *I*, *V*, *V* and *VI* are from the same three leads and show the normal rhythm. The outlines of the auricular beats during the flutter are dotted on the curves where they can be identified. The time marker in this figure is in one-fifth sec..

Fig. 35. Republished from Lewis and Schleiter's paper. The outlines of the auricular waves are dotted upon the curves in leads *II* and *III*. The time marker in this figure is in one-fifth sec..

Fig. 36 and 37. Electrocardiograms accompanied by radial curves in cases of flutter. Showing how the *Aa-Vs* intervals vary when the ventricle responds irregularly.

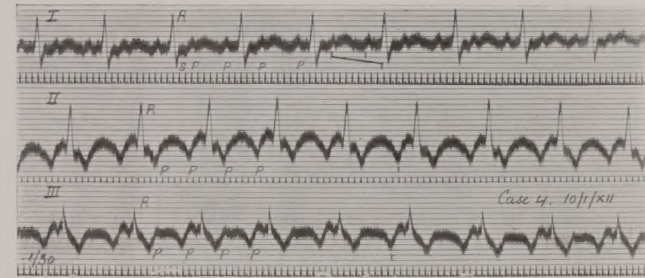


Fig. 28.

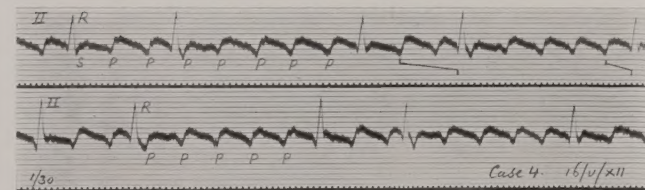


Fig. 29.

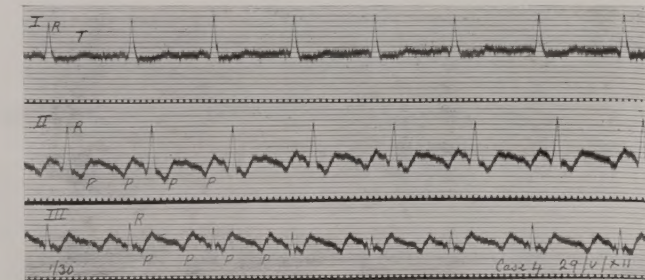


Fig. 30.

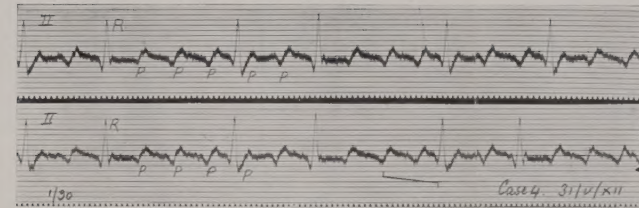


Fig. 31.

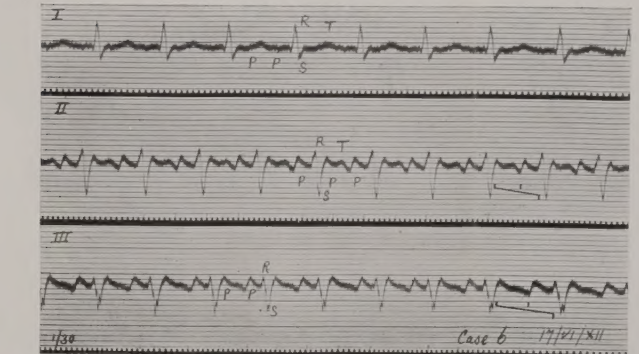


Fig. 32.

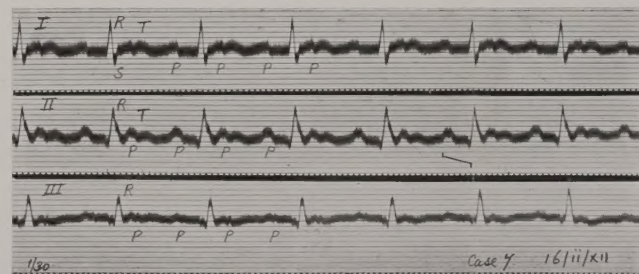


Fig. 33.

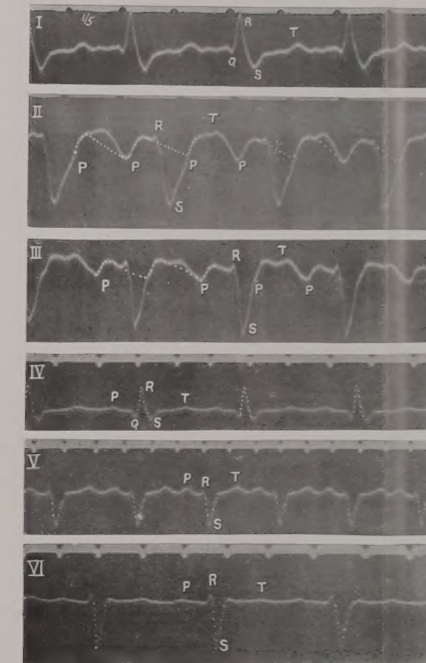


Fig. 34.

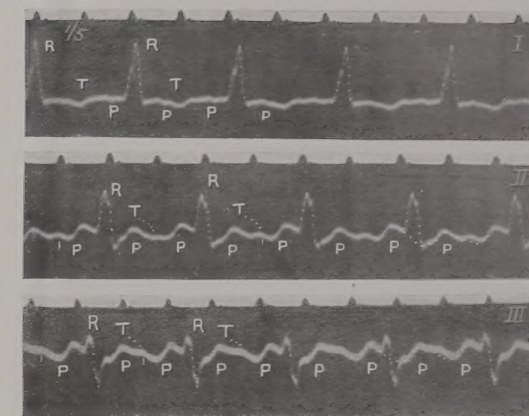


Fig. 35.

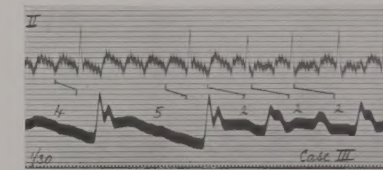


Fig. 36.

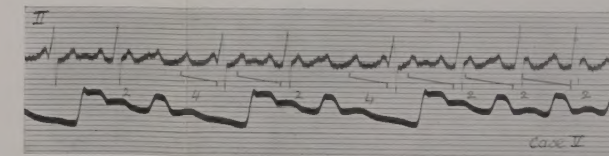


Fig. 37.

CONTENTS.

EXTRASYSTOLE AND THE STAIRCASE PHENOMENON	123
---	-----

BY E. W. GOTELING VINNIS.

(*The Hague.*)

PAROXYSMAL TACHYCARDIA	128
--------------------------------	-----

BY T. STUART HART.

(*New York.*)

(*From the Second Medical Division of the Presbyterian Hospital.*)

OBSERVATIONS ON A CASE PRESENTING A LONG <i>A-C</i> INTERVAL, ASSOCIATED WITH SHORT PAROXYSMS OF TACHYCARDIA ARISING IN THE JUNCTIONAL TISSUES	137
--	-----

BY A. W. FALCONER AND GEORGE DEAN.

(*Aberdeen.*)

ON SIMULTANEOUS RECORDS OF THE HEART SOUNDS AND THE ELECTRO- CARDIOGRAM	147
--	-----

BY GEORGE FAHR.

(*From the Physiological Laboratory of the University of Leyden.*)

OBSERVATIONS UPON A CURIOUS AND NOT UNCOMMON FORM OF EXTREME ACCELERATION OF THE AURICLE. "AURICULAR FLUTTER"	171
--	-----

BY THOMAS LEWIS.

(*Cardiographic Department, University College Hospital
Medical School.*)